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ORIGINAL ARTICLES.

THE RELATION OF ALBUMINURIA TO LIFE ASSURANCE.[1]

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IT is not unusual to hear that medical examiners of life insurance companies have passed candidates who are the subjects of well-established and, it would seem, easily recognized kidney disease; but it may occasion surprise to hear it asserted that a certain number of good risks are lost to companies by the indiscriminate rejection of all cases of albuminuria. This, however, I believe to be strictly true, and it is the object of this paper to point out these, and to discuss the question as to whether it is possible to save them. The importance of such discrimination is manifest, since it will be admitted by every one that no system of life insurance is perfect which does not include all who are essentially healthy, as well as exclude those who are not.

The terms normal or physiological albuminuria, functional, intermittent, cyclical, and alimentary albuminuria have become familiar to every reader of medical literature. They all refer to an albuminuria which is associated with an otherwise apparently healthy organism. No one of them conveys an accurate notion of what is meant, but the term I prefer is *functional* albuminuria.

The words "normal albuminuria" literally mean that the albuminuria itself is normal, which is not intended. The term "intermittent" implies an intermission, which does not always occur, while an albuminuria acknowledged to be abnormal may also be intermittent. The term "cyclical" supposes a periodicity in the intermissions which often occurs, but is by no means constant. Alimentary albuminuria represents a single class of cases in which the albuminuria succeeds the ingestion of food.

Functional albuminuria, on the other hand, simply means that there is somewhere a functional derangement, as the result of which, without structural alteration in the kidney, albumin transudes through its vessel walls along with the normal urine constituents. Of the immediate cause of this I believe it may be safely said we are ignorant, and to speculate upon it is foreign to my present purpose; but I am inclined to believe it is either some quality of the albumin which favors its transudation, or some excess of it in the blood as the result of which it, so to speak, overflows its banks.

I may allude also, in passing, to certain observations of Ralfe, Noel-Paton, and Oliver, as the result of which it is reasonable to suppose that where albuminuria succeeds the ingestion of proteids it is due to an over-stimulation of the red blood-disk destroying office of the liver whereby these bodies are too rapidly destroyed to permit the resulting albumin to be converted into urea, whence again the blood becomes overcharged and the albumin escapes.[1]

For the present we cannot get nearer the truth than this, but this much is now generally admitted, that the person thus affected is essentially healthy. Let me narrate briefly two cases under my observation which illustrate precisely what I mean.

CASE I.—On March 3, 1883, I first examined, at the request of Dr. W. W. Keen, of Philadelphia, the urine of a gentleman, then aged thirty. It had been passed in the morning on rising, and was essentially normal, containing neither albumin, sugar, nor casts. It had 34.5 grammes of urea to the litre. The evening urine contained one-fifteenth of its bulk of albumin but no casts, and there were 21.6 grammes of urea to the litre. Five days later a volumetric examination of the twenty-four hours' fluid discovered 29.8 grammes of urea, the average of two analyses. In addition to the fact that there was no albuminuria on rising, it was also noted that while exercise had the effect of increasing the albumin, massage had not. The most striking symptom was pain in the muscles of his legs, accompanied by a "tired" feeling, in seeking for the cause of which Dr. Keen discovered the albuminuria.

In June, 1884, the patient consulted Prof. T. Grainger Stewart, of Edinburgh, who gave the following opinion: "I have to-day completed the study of ——'s urine. He suffers from intermittent albuminuria, occurring chiefly after food and exertion. He has no evidence of Bright's disease, his urine being of good specific gravity, and containing upward of 400 grains of urea in the twenty-four hours. There is also no trace of sugar. There is no change in the heart or vessels, or in any organ,

[1] Read before the Association of American Physicians, Washington, 1888.

[1] Ralfe: On Some Clinical Relations of Functional Albuminuria, Lancet, 1886, ii. p. 764. Noel-Paton: Relation of Urea-formation to Bile Secretion, Journal of Anatomy and Physiology, October, 1885, January, 1886. Oliver, Thomas: Relation of Urea to Certain Diseased Processes, British Medical Journal, November 27, 1886.

and I regard his albuminuria of the functional kind which has been named 'alimentary albuminuria.'"

At Dr. Stewart's suggestion the patient took a long sea voyage. Six months after his return he came under my care. On January 18, 1886, he brought with him, at his first visit, eight specimens all passed on one day. These I examined with the following result: 7.45 A.M., no albumin; 9 A.M., half an hour after breakfast, one-fifteenth bulk; 10 A.M., an hour after breakfast, one-twentieth to one-fifteenth; 1.30 P.M., after rest, one-twentieth to one-fifteenth; 6.30 P.M., after exercise, one-fifteenth; 8 P.M, after supper, one-fifteenth; 10 P.M. *a trace.* I find also the following note: "Mr. C. has no symptoms, except that he becomes easily tired, but he allows his mind to rest a good deal on his albuminuria, and is in the habit of testing his own urine, and says there is much less albumin than there was, that he feels better when at rest, and on the ocean."

In March, Mr. C. again consulted Dr. Stewart, at Edinburgh. Dr. Stewart reported his condition about the same, "no better, no worse." He recommended a milk diet, suggesting eight ounces of pure milk and an unlimited amount of skimmed milk or buttermilk. On April 4th, under my directions, he began the milk diet, weighing at the time one hundred and twenty-five and three-quarters pounds. He took forty-eight ounces the first day, gradually increasing the amount until, on April 19th, he was taking four quarts a day. This was continued until April 25th, when I ordered a gradual return to a mixed diet. He first lost weight rather rapidly, losing two and a quarter pounds in six days. But as the quantity of milk was increased he gained, and when he returned to a mixed diet he weighed within a quarter of a pound of what he did at the start. At no time during this period of twenty days was the urine free from albumin, and although the proportion was less, yet it is to be remembered that the quantity of liquid ingested was very large, and that, therefore, the absolute quantity in the twenty-four hours was probably in no degree diminished.

From this time on until my last note, dated May 7, 1888, the albumin diminished gradually but irregularly so that more frequently at different times of the day there would be none whatever, being always absent on rising; it would oftener be absent after meals, while the average quantity of all examinations was less. His life is always one of considerable activity, and in summer he is extremely active all day long on a country place. At the last examination, at 9.30, after breakfast there was one-twentieth bulk; at 1 P.M., after exercise but not violent, one-fifth; 3 P.M., after dinner none; at 4.30 P.M., after rest, none; 7 P.M., after driving, a trace; 9.20 P.M., after supper and rest, one-thirtieth. With slight drawbacks his general health steadily improved, and, early in 1887, he married. *Casts were never found.*

As to the determining causes of increased albuminuria, my own study of the case added nothing to the conclusion already arrived at by Prof. Stewart, that food and exercise both tended to increase it, except that possibly exercise exerts a somewhat more potent influence than food.

CASE II., illustrating the class I desire to portray, is that of a gentleman, now forty-one years old. For fifteen years his mother has been passing uric acid gravel. Two maternal uncles died when about fifty-eight years old, having had gout since they were forty. The only suggestion of gout shown by him is an occasional tenderness or stiffness about the small joints, with slight lateral swellings.

From 1880 to 1883 I repeatedly examined his urine, finding nothing abnormal, except a slight hyper-acidity, until August, 1883, when I found a trace of albumin by the picric acid test. On each occasion there had been vague tenderness over the region of the right kidney, probably muscular. He is disposed to headache when constipated, on account of which takes care to keep his bowels free. On November 29, 1883, he called upon me complaining of discomfort, rather than pain, in the region of the right kidney, already referred to, a sensation he found difficult to describe. A portion of the past twenty-fours' urine was acid, had a specific gravity of 1.018, was a pale yellow, and contained neither albumin nor casts.

On January 13th, he was examined for life insurance. The medical examiner after his own examination brought me a specimen of urine, perfectly clear, specific gravity 1.020, in which I discovered a mere trace of albumin by the contact method with picric acid, and by the heat and acid test. On the 18th of the same month, the patient himself brought me a specimen, which also contained a trace of albumin by heat and acid, but which did not respond to nitric acid alone. The quantity on subsidence was decidedly less than that in the urine brought me by the life insurance examiner. On January 20th the urine responded to neither test. *None of these specimens contained casts*, but considerable uric acid appeared some time after the urine was passed.

On January 25th, my patient brought two specimens of urine, one voided in the evening just after tea, the other on rising Saturday morning. Neither contained albumin. On Saturday evening he went to a dinner party, ate freely of rich food, and drank champagne. After dinner he played on the violin for two hours, music requiring much expression and concentration. The urine passed the same night on retiring contained one-tenth bulk of albumin, and that brought to me had a copious sediment of urates. In that of the next morning there was a trace of albumin, but the most careful examination failed to discover casts. Urine passed Sunday evening after a supper of crackers, coffee, and preserves furnished a trace, as also did that of Monday morning. A portion of the twenty-four hours' urine also contained a trace.

Two days later, on rising, the urine was absolutely free from albumin; but after a breakfast of tripe, bread, and milk I found one-tenth its bulk. At 2.15 P.M., having attended a board meeting from 12 to 1.30, there was a mere trace. At 5.30 P.M., dinner and office business intervening, there was no albumin; nor was there any at bedtime.

The next day on rising there was absolutely none. That passed at 10 A.M., the first passed after breakfast consisting of a chop, oatmeal, and cream fol-

lowed by pepsin and a railroad journey of forty miles, was without albumin. After one and a half hours in court testifying, no albumin. After dinner consisting of chicken, bread, milk, and potatoes, none. That passed on returning to Philadelphia contained none. After a tea of oysters and milk-toast there was a trace, and at 12.30, after a visit, also a trace, rather more than after tea.

Such notes as these were made from day to day, and may be regarded as fair samples of the average condition. Always, with the exceptions named, the urine was without albumin on rising, but it was impossible to discover any law followed by the albuminuria at other times. At one time after breakfast, although at home writing quietly, there was one-tenth of albumin; at another there was none. On one day there would be a trace at each examination; at another, although more rarely, there would be none. This important fact was, however, ascertained, although the urine passed on rising contained no albumin, that passed at 9.30 A.M., *but before any food had been taken*, contained one-seventh its bulk. Urine passed at 11.05 A.M. the same day, one and a half hours after breakfast, contained a *trace;* that passed at 2.30 P.M. contained a mere trace; at 4.45 P.M., two hours after dinner, none; and at 6 P.M. none; that passed after tea contained none; at 11.45 P.M., after a lunch of roasted oysters, weak coffee, bread, and a teaspoonful of sherry wine, a trace. The last urine had a specific gravity of 1.030. This day's observations showed that the albuminuria did not, at least, necessarily depend on food; but rather that exercise was responsible for it. At other times, however, after exercise and after food there was no albumin. It was observed that there was almost invariably a tendency to increase after breakfast under any circumstances, almost as invariable as there was absence on rising. Next to the absence of albumin on rising, absence was noted more frequently between 4 and 6 P.M. More commonly there was albumin when the specific gravity was high, yet I note that at least once with specific gravity of 1.036 there was none.

In this case the albumin also became gradually less, and ultimately disappeared. Quite an important observation was this, that the use of "beef-tea" or "beef-essence," as prepared at home, invariably increased the albuminuria.

Contrasting these cases I would call attention to the fact that although in the matter of family history and age the second case would be regarded as the more unfavorable, yet in it the albumin disappeared altogether, while in the first it is still intermittently present.

These cases I regard as illustrating a class in which, although there is albuminuria, its subjects have not renal disease and are insurable risks. Such conclusion is, however, based upon a study of the cases extending over months in the one instance, and years in the other, a study which is seldom, if ever, possible to the ordinary medical examiner. There can be no doubt, however, that could a set of rules be laid down for his guidance the well-trained examiner might save to his company a certain number of risks which are now lost by it. Unfortunately from necessity perhaps, at the present day, the majority of medical examiners are neither well educated nor well trained. Supposing, however, that these important desiderata are attained, as it is reasonable to believe they will at no distant day, are there any conditions which if observed will enable the companies to secure these risks which are now rejected? I believe there are and I submit them to your consideration.

First. It is scarcely necessary to say that the applicant must in all other respects present the signs of good health.

Second. The albuminuria should be unaccompanied by tube casts. However perfect may appear the health of an applicant with albuminuria, the presence of casts in his urine must effectually close the gates of life insurance against him. Casts and albumin conjoined can receive only one interpretation—structural change in the kidney either acute or chronic. Here, however, let me say I have known men with good medical education say that casts were passed in a given specimen when there were none; certain granular aggregations having been mistaken for them.

Third. If the quantity of albumin is large, the applicant should be rejected irrespective of the presence of casts. The term large albuminuria admits, of course, of considerable latitude in its application; but I should say that an albuminuria is large in which the albumin habitually exceeds one-fifth the bulk of the specimen examined.

Fourth. A consideration which goes far toward establishing the functional character of an albuminuria, although not essential to this end, is the absence of albumin on rising in the morning. Nor dare it, of course, be said that such an albuminuria precludes the existence of organic disease. It must be taken in connection with the other considerations mentioned.

A *fifth* point of importance is specific gravity, meaning thereby the specific gravity of the twenty-four hours' urine, or what my friend Dr. Charles W. Purdy, of Chicago, calls the "real" specific gravity. The specific gravity of any portion of the twenty-four hours' urine, or the "apparent" specific gravity, is of limited value, varying greatly with the time of day and relation to meals. If we regard the specific gravity of a normal twenty-four hours' urine (say 50 oz.) as 1.020, the following may be laid down: *albuminuria is least significant when the specific gravity is high*, throwing out, of course, the consideration of sugar. The specific gravity is a direct measure of the solids, which in health are strikingly constant for each twenty-four hours, while the same is true in a less degree of the watery part of the secretion. Hence, the specific gravity of the twenty-four hours' urine is tolerably constant.

In all forms of renal disease the solids are diminished, and in all except acute nephritis and cyanotic induration the specific gravity is lowered. In these two it is lowered because in them the quantity of urine is also markedly less, and, in consequence, the specific gravity is increased to 1.028 and even higher. In acute nephritis the presence of blood may also contribute to such specific gravity. Strictly speaking, acute nephritis may be ignored, because no one ill of it is likely ever to apply for life insurance. In functional albuminuria the specific gravity remains normal. Dr. Purdy even says that his observations in many of these cases show a specific gravity above the normal standard, reaching 1.022 to 1.030.

Whenever, therefore, in addition to the absence of other symptoms and signs alluded to, the "real" specific gravity is above 1.020, another most important fact is in evidence against the presence of organic disease and in favor of the view that the albuminuria is functional. On the other hand, if the real specific gravity of the urine be 1.012, 1.010, 1.008, or even less, as it sometimes is, it would be hazardous to accept such a case of albuminuria, however good may be the apparent health of the applicant and even in the absence of casts. To judge unfavorably from the specific gravity of any portion of the twenty-four hours would be exceedingly unfair to the candidate.

Sixth. The signs of hypertrophy of the left ventricle and of high vascular tension associated with albuminuria are conclusive symptoms of renal disease and should exclude the candidate. High vascular tension may sometimes be shown to exist by the sphygmograph, even when not appreciable to the touch.

Seventh. A highly important consideration is the age of the candidate. Albuminuria is much less apt to be of the functional kind in persons over forty years of age than in those who are younger, especially between the ages of twenty-one and thirty-five. It is questionable whether any person with seemingly functional albuminuria who has reached the age of forty should be accepted unless he has been long under the observation of a competent and conscientious observer.

Eighth. The presence of true gout in any shape precludes admission to life insurance, because gout is always sooner or later followed by interstitial nephritis.

Finally. The retinal symptoms so commonly associated with chronic Bright's disease, although usually late in their appearance, do sometimes form the earliest noted sign of this affection, and whether or not conjoined with albuminuria must effectually exclude the candidate from its advantages.

These, in brief, are the conditions which, if observed, appear to me to enable one to establish conclusively the functional character of an albuminuria, the presence of which should not exclude a candidate from life assurance. They might have been further elaborated, but I prefer to leave them to be discussed as presented. It is to be remembered, however, that observations should extend over a considerable period of time, also that there is reasonable doubt as to whether the requisite knowledge and training are sufficiently general to secure intelligent recognition of the conditions. Take, for example, so simple a matter as examining for tube casts, which may be regarded as almost the easiest sort of microscopical work. Yet, how few medical examiners are competent to make such examinations. Most of them have no microscopes, and many who have them use them so rarely that their opinion whether positive or negative as to the presence of casts cannot be relied upon. On the other hand, there are those who are competent to apply these conditions, and it is reasonable to suppose that this number will gradually increase.

I conclude, therefore, that life assurance associations should recognize a condition of functional albuminuria out of consideration of their own interests, as well as in justice to a class which is coming to be recognized as more numerous as our knowledge increases, and who are blindly excluded from benefits to which they are entitled.

CHRONIC INTERSTITIAL NEPHRITIS AND ANGINA PECTORIS.[1]

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THE subject which I propose to consider in this paper is the relation between certain symptoms referable to the kidneys, and certain other painful and oppressive symptoms relating to the heart.

The former of these two groups of symptoms consists of the occasional presence of albumin in the urine, the occasional occurrence of tube-casts, commonly of a fine granular or hyaline character, and, in general, a low specific gravity of the urine; signs pointing to that form of Bright's disease which is pathologically a chronic interstitial nephritis. The latter group is made up of a feeling of constriction in the chest more or less constantly present, sometimes in moderate degree and sometimes so greatly intensified as to be an acute agony, or an anguish even deeper than that, "a sense of ruin which is worse than pain, a masterful negation and collapse," a feeling of impending dissolution; the condition, in a word, to which the term angina pectoris is commonly applied, and with the emotional as well as the sensory phenomena belonging to that dire disease.

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[1] Read before the Association of American Physicians, Washington, 1888.

Each one of these groups of symptoms is, taken by itself, almost a commonplace of medical experience; but their association with each other is perhaps not very frequently met with, and yet must be occasionally encountered by physicians in general practice. How are they correlated with each other? Is the connection merely an incidental one, so that the anginoid symptoms might have occurred in any given case even had there been no renal disease? Is the condition indicated by the signs referable to the kidneys the direct cause of the cardiac symptoms? Or is there anything in the state of a heart laboring under such symptoms which may directly or indirectly determine albuminuria?

It is well known that some forms of Bright's disease are likely to cause, or at least to be associated with organic changes in the heart, and the manner in which such cardiac changes are brought about has been the subject of much ingenious speculation and able argument, as instanced most recently in the thoughtful and suggestive Middleton-Goldsmith lecture of Professor DaCosta.

It is equally true that an organic change in the heart resulting in dilatation of the organ and general venous stasis may determine secondary disease of the kidneys. But the cases which I have in view would not seem to belong to either of these categories. They may occur without any hypertrophy of the heart ascribable to the renal disease, if it be admitted that the latter can cause such cardiac change, and they may occur also without any such dilatation of the heart as could of itself have produced passive renal congestion. Are both groups of symptoms the results of a common cause acting upon both of the involved organs? I ask the question, but I propose to consider the subject chiefly from a clinical point of view, narrating some of the cases in which I have observed these conjoint symptoms, suggesting perhaps some explanation of them, but trusting that they may receive from others a more complete pathological interpretation than I am myself able to offer.

In the first place I would state that, so far as my reading goes, there is no express mention of a relation between these maladies in any systematic treatise upon the subjects.

Heberden, who first described angina pectoris and gave it the name by which it has been known since his day, states that he had seen nearly a hundred persons suffering with the disease, yet he makes no reference to dropsical symptoms as ever attending it. This he would probably have done had he observed them, though, of course, without associating them with kidney disease, as his observations were anterior to the existence of renal pathology.

In Dr. John Blackall's report of cases of angina pectoris appended to his work on *Dropsies*, which was published before the year 1820, he quotes an account of the case of Lord Clarendon, who died of angina pectoris occurring with symptoms which, as Dr. Blackall says, "rendered probable the existence of some disorder of the urinary organs." These symptoms were great frequency of micturition occurring without pain in the urinary organs themselves, but with intense pain in the left arm. "The circumstances" says Blackall, "have been scarce heard of before, and the causes are not yet understood by any physician."[1] It would seem not unlikely that in this case the morbid state of the urinary organs unattended with pain was chronic interstitial nephritis which is so often accompanied with increased frequency of micturition.

To come to a somewhat later time we find no mention of the combination of the heart and kidney symptoms in Sir John Forbes's masterly article on angina pectoris in the *Cyclopædia of Practical Medicine*.

Professor Gairdner, in his very elaborate and able discussion of angina in Reynolds's *System of Medicine*, merely remarks that "renal disease forms a very serious and often unmanageable complication" of angina;[2] as it certainly does, especially if it give rise to pulmonary œdema; but he does not refer to any pathological connection between the renal and cardiac changes. Nor does any systematic writer, so far as I have been able to ascertain. And yet in several authors there are observations which might have led to the thought of a connection between the two diseases. Thus Gairdner remarks that Lauder Brunton's case in which the value of nitrite of amyl in angina was first made known, "shows a correlation of some kind between angina-like paroxysms and increased arterial tension;" and it is well known that increased arterial tension is common in Bright's disease. Indeed, Brunton himself, in his statement of the therapeutic applications of nitroglycerine, after referring to its use in angina pectoris, says that "it is useful when the heart is weak, or when the tension is abnormally high, as in Bright's disease."[3]

The consideration of the therapeutic fact that the same agent is beneficial in the two diseases, and in the same way apparently—that is, by lessening arterial tension, might suggest a relation of some kind between them. Still nearer the mark is an observation of Strümpell, in his account of the chronic contracted kidney, that "the long-known asthma of renal disease, often termed, without reason, 'uræmic asthma,' does not always have the same origin. It usually depends upon cardiac weakness, and is a genuine cardiac asthma, *corresponding precisely* to angina pectoris in its different

[1] Blackall on Dropsies, p. 255.
[2] Reynolds's, vol. iv. p. 546.
[3] Brunton's Therapeutics, p. 669.

symptoms."[1] But he does not attempt to trace a pathological connection between the diseases.

One case of coexistence of angina pectoris and Bright's disease, closely resembling those I have myself observed, is reported by Dr. Von Russdorf, of Berlin.[2]

I have selected from my note-book the following cases as illustrative of the combination of diseases to which this paper refers:

CASE I.—G. A., female, seventy-one years old, had for more than a year been failing in strength and appetite, becoming pallid in hue and troubled with a feeling of constriction and oppression in the chest, especially referred to the cardiac region. Occasionally there was slight swelling about the ankles, transient in duration, and disappearing entirely at intervals. On auscultation, there was no evidence of pulmonary trouble of any kind, nor did the heart show any sign of valvular disease, but its action was habitually and markedly weak. The urine showed at times slight traces of albumin, which again would entirely disappear, and there were occasional fine granular and hyaline casts—never abundant, and often absent or not discoverable even when a rigid search was made for them. The specific gravity of the urine ranged from 1.012 to 1.015. The diagnosis was that of chronic contracted kidney.

The distressing sense of constriction in the chest increased in degree and persistence, until the 23d of March, 1886, when I was urgently summoned to the patient, and found her suffering with agonizing pain in the region of the heart, extending down the left arm. The expression and color of the face were characteristic of angina pectoris. This pain was promptly relieved by a hypodermatic injection of morphia; but at various times it occurred, though with less violence than at first, while the sense of constriction and oppression in the chest, which had been felt for several months before the severe attack, became more constant. This form of suffering was, however, controlled to a considerable degree by the administration of nitroglycerine in the dose of $\frac{1}{100}$ of a grain three or four times a day. In order to test the value of this remedy, after administering it for two or three days in succession, and until the patient had been apparently relieved entirely of the constrictive pain in the chest, I would withdraw it, when, in a day or sometime in two days, she would request its use again. "Give me my little pills," she would say, and again would experience the desired relief from them. She was provided also with pearls of nitrite of amyl in case of the recurrence of an attack of great severity, and occasionally had recourse to them. And so the contest with two mortal maladies went on, in which, in view of the patient's age and the nature of the diseases, little could be done beyond relieving suffering and endeavoring to promote euthanasia, when, on November 19th, or about seven months and a half after the first acute paroxysm, the patient, on walking from her bed to a chair, sank into it and expired.

In this case the symptoms of angina pectoris were classically marked, as were those of Bright's disease in the form, apparently, of the chronic contracted kidney, as shown by the intermittent occurrence of albumin in the urine, the occasional presence and frequent absence of tube-casts, and the small amount of dropsy. There was no opportunity of making a post-mortem examination.

CASE II.—N. B., sixty-four years old, a merchant by occupation, consulted me in November, 1887, for oppressed breathing and a painful sense of constriction in the chest, extending to the left arm, and felt, in general, only when walking, especially when walking up hill or up stairs. Auscultation showed no valvular change in the heart, but its action was weak, and at that time there was no sign of pulmonary disease. Examination of the urine gave a negative response as to the existence of disease in the kidneys, but a few weeks later it showed a cloudy trace of albumin and scanty hyaline and fine granular casts. Later repeated examinations showed sometimes the presence and sometimes the absence of these signs, as is common in chronic interstitial nephritis. The patient was troubled with constipation, and was often imprudent in eating; and the regulation of the bowels, together with restriction for a time to a milk diet, produced a marked improvement in the breathing and a lessening of the constrictive pain in walking. At the same time he was put upon the use of $\frac{1}{20}$ of a grain of arseniate of iron three times a day. In January, 1888, anasarca appeared in the lower limbs as well as slight effusion in the pleural sacs and patches of pulmonary œdema. These symptoms abated under the influence of digitalis and tincture of the chloride of iron.

The weather in Baltimore being at that season very unfavorable for one in the patient's condition, he went, by my advice, to the milder climate of Thomasville, Georgia, where he consulted Professor John T. Metcalfe. This gentleman wrote to me that he detected in the patient's lungs the presence still of some œdema in scattered spots, and he stated that he had frequently met with this patchy sort of œdema in Bright's disease, and thought it somewhat characteristic of the malady. I believe it often is so, and perhaps it is analogous to another localized form of dropsy occurring in the same disease, that, viz., in the lower eyelids. In April the patient returned from Georgia, and in the drier and warmer weather of the spring and summer he has somewhat improved; the effusion in the pleural cavities, the lungs, and the lower extremities has disappeared; but the chemical and microscopical signs of renal disease still exist, and he still suffers occasionally with attacks of angina. On July 19, 1888, albumin was present in the urine to the amount of two per cent. by volume; there were a few hyaline casts, and the specific gravity was 1.015. On August 18th, there was three per cent. of albumin by volume, no casts, and a specific gravity of 1.011.

In this case, again, the coexistence of the symptoms of angina pectoris and of chronic contracted kidney is plainly shown.

[1] Strümpell's Text-book, Amer. edit., p. 808.
[2] Deutsche Klinik, vol. xviii. p. 375.

CASE III.—J. W., sixty-three years of age, a physician by profession, was able to appreciate the full significance of all the symptoms belonging to his mortal maladies and to meet them with fortitude. He had been failing in health and growing weak and anæmic for many months, when in February, 1888, I was invited by his attendant physician to see him in consultation. The most prominent symptoms then existing were great oppression and sense of weight in the cardiac region, with attacks of acute pain in the same situation so severe as to render the hypodermatic use of morphia necessary, and also the inhalation of nitrite of amyl, from which temporary relief was gotten. The administration of nitroglycerine was without effect. Dyspnœa was frequently urgent, but was not dependent upon any change in the lungs or heart discoverable by auscultation. Anasarca in moderate degree sometimes appeared in the lower limbs, but was transient in duration. The urine was examined many times, often presenting nothing abnormal, but occasionally showing slight cloudiness when tested for albumin, and sparse hyaline casts under the microscope. Yet the presence of these at all, though infrequent, taken in connection with the character of the breathing and the anasarca, pointed to the existence of chronic contracted kidney, while the breast pang showed angina pectoris. No remedies stayed the progress of his organic disease; but little and only temporary relief could be given to the attendant pain and difficult breathing, and on the 8th of April he died.

At the *post-mortem* examination the kidneys were found to be diminished in size, chiefly at the expense of the cortical portion, and their capsules somewhat adherent, though not to so great an extent as is usual in a more advanced stage of atrophic change. The heart was distended by coagula, giving it the appearance of being enlarged before it was opened, but the ventricular walls were attenuated and large fibroid patches were found under the endocardium of the left ventricle. The coronary arteries were both in a state of fibroid degeneration; the right artery being completely occluded by softened fibrous and atheromatous material, and the left so much obstructed that it was only slightly pervious. This degree of obstruction must necessarily have caused great cardiac ischæmia. An aneurismal dilatation of the aorta of the size of a large egg, with atheromatous walls was found just below the cœliac axis.

Of the three cases here reported, in the first no post-mortem examination could be made. The second is that of a patient still living. In the third the diagnosis of the condition of the kidneys made in life was confirmed by the post-mortem examination, which also showed changes in the heart not positively diagnosticable, but entirely in accordance with the cardiac symptoms observed, and perfectly explaining them.

It seems a fairly probable inference that inasmuch as Cases I. and II. corresponded in the combination of their symptoms closely with Case III., the same or closely similar changes to those found in Case III. existed in Case I., and exist now in Case II.

There would certainly be no reason for regarding the changes in the heart belonging to Case III., and probably belonging also to Case I. and II., as in any way causative of the diseased state of the kidneys. And, on the other hand, there would be no reason for regarding the kidney disease in Case III. as productive of the cardiac symptoms through uræmic poisoning or any merely functional disturbance, when positive organic change fully accounting for the cardiac symptoms was found. If uræmia acting on the heart ganglia were the source of trouble in the same way that it occasions such disturbances as convulsions and coma by its influence on other nervous centres, then these heart symptoms might be expected to be as frequent as are the cerebral symptoms of Bright's disease, which they certainly are not. Nor, again, is it easy to see how the kidney disease could have been the direct cause of the organic change in the heart and the symptoms due to it. Bright's disease may, it is true, give rise to endocardial inflammation and to consequent changes in the cardiac valves; but in the cases referred to no such changes existed, and, therefore, the kidney disease could not have held a causal relation to the angina by producing such valvular lesions.

The morbid changes most commonly found in connection with angina pectoris are obstruction of the coronary arteries, insufficiency of the aortic valves, or rigidity of the aorta, possibly interfering with the filling of the coronaries, and fatty or fibroid degeneration of the heart. Any one of these pathological conditions may give rise to cardiac ischæmia, which is most probably the immediate cause on which the angina depends. Cardiac ischæmia is more directly brought about by obstructive disease of the coronaries than by any other lesion, though atheroma or sclerosis of the aorta may accomplish the same result, as may also endo-arteritis or peri-arteritis of the heart arterioles by preventing sufficient blood from entering the myocardium, even though there may be no coronary obstruction. According to the statistics of Sir John Forbes, coronary disease was found in seventeen out of forty-five cases, or more than one-third.[1] Lussana gives twenty-one cases of coronary obstruction in thirty-six fatal cases of angina;[2] and Liegeois, whose researches are later and probably comprise a larger number of cases, states that three-fourths of all cases may be attributed to atheroma or sclerosis of the coronaries or aorta, or both.[3]

Now all these are degenerative changes, and ap-

[1] Cyclopædia of Practice of Medicine, vol. i. p. 107.
[2] Virchow's Handbuch, vol. ii. p. 422.
[3] Bull. Med. des Vosges.

parently they cannot be directly attributable to any renal disease. It would seem more probable, therefore, that the change in the heart giving rise to the symptoms of angina is, as is ably maintained by Professor Da Costa in reference to the cardiac hypertrophy of Bright's disease, not the direct consequence of that disease, but a part of a general degenerative change, of which one expression is manifested in the heart and another in the kidney. As Russdorf remarks in the report of his case, "the renal change is the last stage of a slow process of disease, of which the beginning was in the heart."

And, perhaps, if we are inclined to believe with Dr. Da Costa and Dr. Longstreth, that "it is in the nervous system that we are to search for the original source of the malady," we may think that some further probability is lent to this view by the pathological fact of the increased arterial tension found both in Bright's disease and in angina pectoris, and due to a change in the vaso-motor system, and possibly in the cervical ganglion, and the further therapeutic fact that nitroglycerine, which lessens this tension, has proved beneficial in both affections.

IMAGINARY LINGUAL ULCERATION.[1]

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"IMAGINARY lingual ulceration," with its demoralizing pain and indefinite terrors, is very real to the sufferer, while to the physician the almost utter futility of his present therapeutic resources for its relief renders the affection one of more than passing interest and importance.

These two reasons alone, even if others did not exist, would justify my bringing the subject briefly before you for discussion, which I now do, in the hope and belief that light may be shed, especially upon the vexed questions of etiology and therapeusis.

"Imaginary lingual ulceration" — recently so named by Verneuil—although a comparatively rare affection, is doubtless well known by some name or under some form to many of you. It presents no complex symptoms; its local lesions, if any exist, are often indefinite; still the diagnosis usually admits of no doubt, and the clinical history, taken alone, is distinctive.

A patient, male or female, as the case may be, for the affection occurs with equal frequency in the sexes, and from thirty-five to fifty years of age, presents himself complaining of intense pain, usually of a lancinating or darting character, limited to one-half of the tongue, usually the left, and extending from its base, just opposite and below the tonsil forward to its tip, or the pain may be more circumscribed and limited to the junction of the posterior with the middle third of the organ, at or near the location of the posterior lenticular papilla. It is more or less intermittent, rarely constant; is aggravated by movements of the tongue, although these are free; is often increased by the act of mastication and excited by hot or irritating articles of food. Its effect upon the patient is demoralizing, his imagination plays him false, his terrors are aroused, and injudicious medical or surgical interference and advice, instead of allaying both mental and physical disturbance, not infrequently excite them; the fear of cancer, and the belief, which remains unshaken, that ulceration actually exists, constitute an integral part and important factor in this curious affection.

Such patients, however, are not invariably hypochondriacal, as has been asserted; on the contrary, the affection is met with in those who present no evidences of a neurotic or even nervous temperament; it is occasionally seen in individuals possessing and presenting all the signs of robust health, and the mistake has been made, and is made to-day, of ascribing alone to the imaginative fears of the patient what actually exists as an undoubted, if not always demonstrable, physical and painful fact.

Subjective symptoms, other than the pain as described and occasionally slight salivation, if it be excessive, do not exist.

Careful physical examination reveals but little or nothing to guide us to a diagnosis, especially if the tongue, at its affected point, has not been tampered with by local and irritant applications. If drawn forth from the mouth and extended to the opposite side from that on which the local point of pain is complained of, it usually presents a normal appearance; the surgeon's attention being directed, as it always is, by the patient to a point on the line of demarcation between the roughened epithelium of the dorsum of the organ and the smooth mucous membrane below, a lateral line, which is marked near the tonsil by the invariable presence of a few delicate papillæ. These will be found of natural number, size, and color, subject to normal variations in different individuals. If irritation has been caused, these papillæ will be seen swollen, reddened, and associated with lateral inflammatory enlargement of the tongue to a greater or less extent, pain is increased and constantly aggravated by the swollen tongue being impinged upon by the teeth during mastication. Lesions of a catarrhal nature, which commonly coexist in naso-pharynx, pharynx, and perhaps larynx or tonsils, are not of such a character or grade, as a rule, as to influence a part so remote and indirectly connected, nor are they such as to excite a plausible suspicion as to a possible reflex character of the tongue disturbance.

Such is a brief physical sketch of the affection

[1] A paper read at the Tenth Annual Meeting of the American Laryngological Association.

which we have named "imaginary lingual ulceration." A fanciful term, perhaps, as ulceration rarely if ever exists, but certainly descriptive enough from our, and truthfully exact from the patient's, point of view, to warrant its being retained.

In passing to the question of causation, we are at once involved in a multitude of hypotheses, bewildered by more or less plausible explanations, and confronted by a variety of views. No unanimity of opinion exists. From the little that is found in literature concerning the affection, I select the following, first recording my own opinion, based upon experience, that to regard it and to dismiss it at once as a fanciful conceit on the part of the patient, is a grave mistake, and that relief of the real suffering that undoubtedly often exists—granted that this may be in part mental—is not and cannot be attained by such a course.

Verneuil (*Acad. de Méd. de Paris*, July 27 and October 4, 1887), and I believe Labbé, regard the disease as a purely neuralgic affection of the tongue, and compare it to an affection of the breast, described by Velpeau under the name of imaginary tumor of the breast, which, like the above-named disease, is also characterized by intense local pain without any appreciable lesion.

Fournier, Lollé, Hardy, Luys, and Magitot all agree to regard the symptoms as hypochondriacal.

Poyet (*Journ. des Conn. Méd.*, October 20, 1887), from whom I quote, says that the delusion may almost always be referred to one of four causes. In the first class of cases there is a neuralgia of the tongue, and the pain leads the sufferer to believe that ulceration exists. In the second class are patients who have read or heard of some person afflicted with cancer, and have been led thereby to examine their own tongues. They notice, perhaps, a somewhat prominent papilla, and their fears lead them to the conclusion that it is the edge of an ulcer or a commencing cancer. Others are hypochondriacs, pure and simple, and are so convinced that there is an ulcer present, that no amount of argument or demonstration can convince them of their error. Finally, certain patients, with lingual psoriasis, discover the white patch on their tongue and think that ulceration is present.

These old-time views, representing but one side of the question, I present, for the moment, without comment.

Albert (*Wiener med. Presse*, Nos. 1 to 6, 1885) ascribes the pain to the presence of "irritable papillæ in the *regio foliata*." But cases are certainly met with in which none of these hypotheses covers the diagnostic ground. Cases such as I have described above, not neurotic, not imaginative, of good general physical condition, cases in which all possible or probable sources of local irritation have been removed, such as decayed, irregular, or ragged teeth which press upon the tongue, teeth filled with amalgam, which possibly chemically affect it, diseases of the gums, which have been claimed by recent writers to be efficient cause for reflex neuroses of distant parts, but still pain persists. *Why*, is the question that we must ask ourselves. Are general causes at work of which we at present know nothing? Can a rheumatic or gouty diathesis with such local manifestations be at fault? Doubtless such cases exist. I have seen and treated them successfully; but they are limited in number. Must we then be driven, by our etiological uncertainty, into that indefinite and unsatisfactory but comfortable diagnostic haven of "neuralgia?"

I have alluded to the common plan of treatment; the patient is classed as hypochondriacal, nervous, fanciful; his sufferings regarded as unreal and possessing actuality in his imagination alone, and as a *placebo* local applications of a caustic nature are made to the lingual papillæ, whether these be enlarged or not, in the hope of curing the supposed growth. The results are such as to increase rather than alleviate pain, and only serve to confirm the patient in his delusion, should this exist. Still more, these results of injudicious treatment may pass, as I have seen, beyond the limit of mere irritation into a stage of actual ulceration, suspicious induration, and dangerous extension, with glandular involvement.

To such a vulnerable organ as the tongue, the danger of such a course cannot be overestimated, nor its adoption too strongly condemned. That it is unscientific and ineffectual requires no demonstration.

Applications of cocaine or of aconite sometimes succeed, often fail; and the same is true of the various treatments with which we are familiar, usually pursued in neuralgias of other parts of the body. Electricity is usually of service. Hypodermatic injections of analgesic drugs have been recommended. More radical procedures have their advocates in cases in which milder means have failed. Cauterization with the thermo-cautery and destruction of the calciform papillæ sometimes, not always, found upon the tongue in such cases. Poyet recommends such a course, or some other painful application, solely for its moral effect; the danger and futility of such a procedure we have seen.

Neurotomy must be discarded, not alone on account of the difficulty attending such an operation in this particular locality, but mainly because of the impossibility of proving whether the lingual or the glosso-pharyngeal nerve is the one at fault.

While thus criticising my own treatment, with its few successes and many failures, and reviewing that of others, I regret that experience has taught me nothing that is uniformly successful in its results to recommend, and my intention has been, in bringing this rare and curious affection before you for discus-

sion, to place it in what I regard as its true light and to profit by your therapeutical experience in its treatment.

Ample room exists to-day for improvement in our methods, and increase in our means directed toward this end.

TWO CASES OF EPILEPSIA PROCURSIVA.

BY H. A. HARE, M.D.

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RUNNING epilepsy is so rarely seen even by neurologists that the following cases may be of interest and are certainly worthy of record:

CASE I. was as follows: Girl, aged eight years, the child of healthy parents, was brought to me by her mother who stated that the child would give a scream and then suddenly dart out of the house into the crowded street and run one or two hundred yards, when she would return to herself dazed and unable to find her home, rather because of her surprise at her new surroundings than an absolute lack of intelligence. At the time I saw the case the attacks were so frequent as to prevent the mother from performing her work, because of the constant watching necessary. In other words, the attacks occurred several times a day. If held so that escape was impossible, the girl struggled to get away, but there were no fits of the ordinary character at any time. There was never any coma after the attacks, no incontinence of urine or feces. There was no history of any cause of the disease so far as could be discovered. The mother was told to bring the child back in one week, but failed to do so and all trace was lost of the case.

CASE II. is as follows: Jesse ——, aged fourteen years, is a strong and apparently healthy boy, of healthy parents. He is a remarkably well-built and manly looking fellow of more than the usual height and weight and is well proportioned. His complexion and color are good and he is notorious at school for his brightness and acuteness in learning his lessons. He is not one of those typically "bright boys" who look as if highly strung, but is perfectly normal so far as his appearance is concerned. He was entirely devoid of any morbid condition until just one year ago, when the first fit took place. The history of his life gives no record which can be, in any way, considered as a cause of his present trouble, save that when he was about six years of age he was struck on the back of the head by another boy who was much larger than himself with a heavy club. No unconsciousness was produced, but blood flowed freely from the wound, the scar of which is still to be seen about two centimetres downward and to the right of the occipital protuberance. No secondary effects ensued from this stroke, but exactly two years ago he was struck while at play on the head within two centimetres of the former scar. The blow felled him to the ground and produced unconsciousness lasting for some minutes.

One year ago, almost on the anniversary of this last blow, he was down in the cellar of his father's house and suddenly rushed up stairs and sat on the kitchen table, muttering, and moving his legs, and apparently unconscious of his surroundings. There was no coma, nor headache after this attack and he seemed perfectly well. The other attacks have since occurred either once or twice a week or even less frequently at certain times. When they occur in the daytime they invariably consist in a sudden run across the room ending sometimes in a fall to the floor, but there are no spasms when he falls. One week ago he was sent to a store for some nails. He entered, asked for the nails, and then suddenly darted out into the street, throwing his money away as he ran. The distance traversed before he was caught was about one hundred yards. When stopped by the storekeeper who followed him, he strove to escape, but a moment after regained consciousness and was absolutely ignorant of how he reached his new surroundings.

Up to this time the writer had never seen him in an attack but on the day this was written, while he was being taken up stairs in the University Hospital in order to be shown to the class, he was seized with a paroxysm in which the following symptoms were noted. The head was drawn slightly to the left, the right shoulder thrown forward and upward and he assumed the position of one about to start on a foot race. He was seized instantly but used the utmost effort to escape, the efforts being ineffectual chiefly because they were lacking in intelligence, but the movements of the legs, although he was held, were running movements finally deteriorating into struggles such as those of an angry child trying to break away from a parent. The entire attack was accompanied by muttering and at the beginning of it the saliva poured from his mouth. The aura in this attack was distinctly visual and consisted in total blindness. Indeed, at the first part of the attack he groped blindly about for something to clutch. This condition of blindness has formed the aura in every fit, so far as can be learned from the boy and his parents. There was no incontinence of urine or feces. The transient stage from unconsciousness to intellection was but momentary and could not have lasted over sixty seconds. The mental clouding after the attack was so slight and transient as to be scarcely noticeable. He has had several nocturnal attacks consisting in turning and twisting in the bed with muttering but no running movements. An interesting fact in regard to the case is that while ten grains of bromide of potash three times a day failed to control the attacks, antifebrin in doses of five grains three times a day produced an intermission of one week which might have been much longer had not the fright of going before a class precipitated an attack.

Unfortunately this case, like its predecessor, promised to return for an ocular examination but failed to appear, and frequent requests have been so persistently disregarded that I have given up all hope of obtaining it as the parents do not seem to regard the disease as being more than a slight annoyance.

CHEYNE-STOKES RESPIRATION.

A Report of Three Cases, with Remarks.

BY WILLIAM O. STILLMAN, A.M., M.D.,
OF ALBANY, NEW YORK.

CASE I.—On December 11th last, I received a telephone message to call upon Mr. C. S., white, married, American-born, aged forty-seven. About 5.30 that same morning, while engaged in wheeling a loaded wheelbarrow on a scaffolding, a portion of the structure fell with him, and caused the barrow to fall upon him striking him in the abdomen. The fall was not over three feet, and he was able to pick himself up almost immediately and extricate himself and his load. Despite considerable shock, he was able to work until late in the afternoon. About 3 P. M. he complained of headache and nausea, grew faint, and for a couple of hours felt unable to attend to his duties, but succeeded in walking to his home, a quarter of a mile away. I saw him about 7 P. M., and found him lying in bed; his mind was somewhat confused, his pulse was about 95, rather hard and full, and his respirations of the Cheyne-Stokes description; the period of breathing and of apnœa occupying the same intervals, some twenty to twenty-five seconds, the number of respirations during each series being fifteen to twenty. The pupil was contracted (slightly) during the period of rest, but dilated during the respiratory paroxysm. Muscular power and movement of every description (arms, limbs, tongue, head, and trunk) were retained.

On being roused he complained bitterly of pain in the epigastrium, where he claimed the barrow struck him, and also of bruises on his legs and right hip. He was able to give an account, interrupted by lapses from which it was necessary to rouse him, of his accident and subsequent conduct. His head (the whole of it he claimed) ached slightly, but he was not conscious of having hit it, nor was any bruise of the scalp discernible. His kidneys worked freely, and there appeared no indications of uræmia, and he had no rigor.

The following day his condition was much the same, except that the peculiar respiration was more marked and the apnœic intervals longer. He partook of nourishment (there was no nausea or vomiting), his bowels were moved freely, his mind was more sluggish, but he did not recognize his wife, although able to answer questions intelligently. At the end of about eight days the Cheyne-Stokes respiration had gradually disappeared, and his condition was much improved. At the end of seven weeks he was able to be out of doors, and at this time (February 26, 1888) he is at work,[1] though feeling somewhat weak and "short of wind." He still suffers a little pain in the epigastrium, and claims a diminished power in the right hand and leg. This last was scarcely noticeable during the first week in bed, and was not a marked symptom at any time. His heart is quite weak in its impulse, evidently from excessive use of tobacco, and the pulse is very compressible. There is no indication of atheroma or valvular disease. Has always been thin and not vigorous. I have presented the symptoms in the order in which they claimed attention. The diagnosis was serous effusion particularly affecting the base and anterior vertex of the brain; but I am inclined to think that there was a small hemorrhage in the vicinity of the left ascending frontal convolution.

[1] He is now, August 18, 1888, in his usual health.

CASE II.—On December 31, 1887, I was called to see Mrs. A. U. L., white, married, American-born, aged about seventy-six. She was unconscious, and presented the unmistakable symptoms and history of cerebral hemorrhage; there being complete loss of power in right arm and leg. The medulla was evidently already affected, the breathing puffy. Cheyne-Stokes respiration was present, the periods of breathing and apnœa being about equally divided in regard to time, and the respiratory series being about thirty each time in number. The pulse beats numbered about 84, and remained about the same during my call. I had previously attended Mrs. L. for heart difficulty, there being marked evidence of fatty degeneration (atheromatous arteries, arcus senilis, attacks of difficult breathing, etc.). There seemed to be no marked valvular trouble. She had also suffered from a slight cerebral apoplexy a few weeks previous, with a very slight disturbance of motility on the side later affected. She died on the morning of January 3, 1888, there having been no particular change in her general condition, except that the intervals between the breathing spells gradually lengthened, the respiration became more stertorous, and there was a progressive loss of strength.

CASE III.—Early on the morning of January 1, 1888, I was summoned in consultation in the case of F. C. F., male, white, unmarried, American-born, aged twenty-seven. The patient had been in apparent health the day before, when, although somewhat inactive, he had walked some distance, played upon a piano, and seemed to be in a cheerful frame of mind. About 11 o'clock in the evening he had complained to a friend of severe pain in the back of his head; a physician was called and a small amount of anodyne administered. He retired between 11 and 12 P. M. At 3 A. M. his room-mate was aroused by his heavy breathing and he was found to be unconscious. The family physician was at once called, and at 5 o'clock I was sent for. Subsequently the physician who had first seen him arrived. Cheyne-Stokes respiration was well marked when the family physician arrived, and continued so up to the time of death, which occurred at about 10 o'clock the same morning. When I first saw him the interval of apnœa was longer than the breathing period, and this condition continued with slight fluctuations and a tendency toward increased apnœa, until his death. His extremities were cold, his pulse rather small but of somewhat increased tension, and frequently the pupils were strongly contracted and varied but slightly in calibre; he was completely unconscious. There were no indications of organic heart disease, but the heart's action was very feeble. He belonged to a somewhat neurotic family, was slight in build, and was an excessive cigarette-smoker. A diagnosis of cerebral meningitis was made and concurred in by all present. Subsequently it was learned that he

had received a sharp blow on the head by running against a telegraph pole while trying to catch a train the day before he was taken ill.

I have not attempted to give the symptoms or histories of these cases with any great minuteness, as that is not necessary for my purpose, which is simply to call attention to the accepted explanation of the causes of Cheyne-Stokes respiration. It seems to me that the classical theory of its causation is defective in certain respects, which I will explain later.

Dr. Cheyne first called attention to this peculiar alteration of the rhythm of respiration in 1818, and described it with great accuracy. In his case it was associated with fatty disease of the heart. Little attention was paid to the symptom until 1846. Dr. Stokes urged its significance as a sign of fatty degeneration of the heart, believing that it was a pathognomonic symptom of this affection, and that its presence indicated a fatal and speedy termination. Dr. Seaton Reid soon demonstrated that it might depend on valvular incompetency only, and Dr. Hayden has shown that it is probably present in about one-third of the cases of fatty degeneration of the heart (presumably those resulting in death are meant). He considered that atheroma of the aorta was the cause of its presence.

Later observers, with wider opportunities, have shown that this symptom is occasionally found in a considerable variety of diseases, relatively most often (it is asserted) in those diseases of the brain which give rise to compression in the neighborhood of the medulla oblongata. Thus it is found in cerebral hemorrhage, exudation, and tumors; it also occurs in uræmia, and in various cardiac diseases, including fatty degeneration, sclerosis of the coronary arteries, stenosis of the aortic or mitral valves; and may complicate abscess and congestion of the brain. Fräntzel, Leube, and Bernheim have reported cases in which it appeared after the administration of morphine; Biot after the use of bromide of potassium; and O'Neill after chloral. Fräntzel's case furnished Traube with the data for his classical remarks on the *rationale* of its causation. His theory is, that lack of arterial blood supply to the respiratory tract in the medulla (a characteristic common to these affections it is claimed) diminishes its irritability to such an extent that a much larger accumulation of carbonic acid in the blood than usual becomes necessary to excite the inspiratory stimulus, and that thus an interval occurs which is compensated for by increased action.

Traube's theory has two great faults; it does not show why the Cheyne-Stakes respiration should not be present in all cases which at times may produce it, and it does not explain why spasmodic compensatory activity of respiration should occur only in these cases of carbonic acid poisoning and under these special circumstances. Filehne, recognizing these deficiencies, extended Traube's hypothesis, and formulated the commonly accepted theory of its causation. He claimed that the vaso-motor centre must be more easily excited than the respiratory centre, and that the symptoms were caused as follows: "At the end of the respiratory pause there is a large disappearance of oxygen from the blood, carbonic acid has accumulated, the vaso-motor centre is thereby stimulated, and the arteries (the cerebral artery among the rest) at once contract; this produces a gradually increased anæmia of the respiratory centre, and inspiration becomes deeper; this, however, supplies the wanted oxygen to the blood; the arterial spasm is relieved, the anæmia of the respiratory centre passes off, and with it the exaggerated impulse to respiration and breathing once more becomes superficial," and with the cessation of the arterial spasm the stage of apnœa is again reached.

Filehne's theory is very ingenious, but I do not see that this explanation has relieved us from our embarrassment. First, he claims increased vaso-motor irritability. Does not this exist normally, as much as it can be demonstrated to exist in these special cases? Does not the sympathetic system (the heart being under its control), as a rule, persist longer in a condition of irritability than the voluntary system, or the semi-voluntary, which controls the lungs (it being the voluntary muscles which are concerned in respiration)? As an instance, the heart of a person asphyxiated by hanging continues to beat for some time after *efforts* at respiration have ceased. Second. He does not explain why a special cause for the symptom exists in some cases and not in others of the same disease. Why should not all cases of cerebral apoplexy or opium poisoning exhibit the Cheyne-Stokes respiration? It seems to me that the missing link, or rather the starting-point in the chain of causation is found in the equilibrium between the respiration and circulation being always disturbed by a relatively weak heart. In all the early cases cardiac degeneration or incompetency was noted. In all of those which I have been able to study personally or through notes, the heart has seemed to be the disturbing factor, even when the disease which has produced the special illness affected other parts. Cerebral hemorrhage, with which this variety of respiration is specially associated, naturally suggests a degenerated vascular system. Since the heart is found to be affected in so large a number of cases of this description, does not this fact offer a reasonable explanation of why certain cases of drug exhibition may or may not show it?

Without discussing the cases which I have instanced in detail, I may say that I believe that they all sustain this view of the situation, although two of them still further disprove the early claim of fatty degeneration of the heart being necessarily present. They seem also interesting as illustrating the range

of age and disease which may furnish this still not fully understood symptom. I am not aware that any one has hitherto pointed out this apparent connection between a *merely weak heart* and the Cheyne-Stokes respiration.

MEDICAL PROGRESS.

The Mortality of Abdominal Section.—At a late meeting of the Royal Medical and Chirurgical Society, MR. W. A. MEREDITH (*Lancet*, October 27, 1888) read a paper, the purpose of which was to draw attention to certain points affecting the present death-rate after abdominal section, and with this view operations for ovarian growths were chosen, as being to some extent representative of the entire subject under consideration. As a basis for the inquiry, the results of the author's own work in this connection, amounting to 126 operations, were examined with regard to the chief causes influencing the mortality, special reference being made to the ten deaths which occurred in the series of 104 completed ovariotomies. All the operations were performed with strict antiseptic precautions, including the use of the carbolized spray. He was now less inclined than formerly to look upon the latter as an absolute essential to the safeguards of antiseptic abdominal surgery, but he still considered it valuable as the most convenient and effectual means of antiseptic irrigation at our disposal when dealing with the peritoneal cavity. Both ovaries were removed in seventeen cases, two of which terminated fatally, but in neither instance was the result in any way attributable to the removal of the second ovary. Complete enucleation was performed in five cases, all successful, the resulting rent in the broad ligament being closed by means of a continuous silk suture. He advocated the use of the drainage tube in all cases of ruptured or inflamed cysts, in any case in which irritating or septic fluid had escaped into the peritoneum, in all operations complicated by serious injury to bowel or urinary bladder, in every instance in which washing out had been resorted to, and in all cases of severe operation in middle-aged or elderly women. For washing out the peritoneum, where necessary, he advocated the use of plain, recently boiled water, cooled as required by the addition of a saturated solution of boric acid.

Discussing the mortality, he was inclined to believe that in a large series of cases the average death-rate would never be less than five or six per cent. Of the ten deaths in his series, two were from septicæmia, one from intestinal obstruction, one from hemorrhage, one from dysenteric diarrhœa, two from chest complications, and three from exhaustion; three of these deaths were from preventable causes. Twelve successful operations for the removal of the diseased uterine appendages were next reviewed. In seven patients, in whom both appendages were removed, complete arrest of menstruation followed. He had found that in cases of chronic ovarian mischief about ninety per cent. were the subjects of obstinate constipation, and in them tonic purgative treatment, by lessening pelvic congestion and preventing the formation of scybala, was followed by marked relief of symptoms. Included in a series of ten exploratory operations were two incomplete ovariotomies. Both patients had been tapped, and the death of one was directly due to the difficulties encountered in the attempt to separate universal adhesions which had resulted from this treatment.

In conclusion, it was shown that the increased success of abdominal section for ovarian disease during the past ten years was chiefly attributable to the diminution in the number of deaths from septicæmia. For purposes of comparison he took three successive groups of operations, which represented three separate periods during the last twelve years, and gave their average death-rate from septicæmia alone. In 100 consecutive cases taken from Sir Spencer Wells's work, when the clamp was used and before the employment of antiseptics, the mortality was ten, or one to every ten patients. In 150 cases recorded by Mr. Thornton in the sixty-fourth volume of the *Medical and Chirurgical Transactions*, which were treated antiseptically and with intra-peritoneal ligature of the pedicle, there were five deaths, or one to every thirty patients. In his own series of 104 cases there were two deaths, or one to every fifty-two patients. The chief factors which had contributed to this increased success, he thought, were as follows: (1) The general adoption of the intra-peritoneal treatment of the ovarian pedicle; (2) the application of the antiseptic system to abdominal surgery; (3) the gradual abandonment of the practice of tapping adbominal cysts; (4) the increase in our knowledge respecting the proper use and management of the drainage tube; (5) the recent introduction of the plan of freely washing out the peritoneal cavity in cases complicated by the extravasation of blood or other fluid.

MR. ALBAN DORAN had seen great advantages follow both the antiseptic and the non-antiseptic systems of treatment. He thus summed up the influences which he thought contributed to lessening the mortality after ovariotomy: The skill and experience of the operator; early operation; a good hospital and efficient nurses, amongst the latter emulation being a great factor in favor of good results; the use of antiseptics, not only for their own value, but because they drilled the operator into habits of forethought; drainage, when the tube was frequently pumped out; and gently flushing the peritoneum with a great bulk of warm fluid, which checked hemorrhage, washed out clots, and prevented shock by keeping the peritoneum warm. He condemned the use of a ward nail-brush, which he believed to be a frequent carrier of infection.

MR. KNOWSLEY THORNTON objected to stitching up the rent in the peritoneal capsule after enucleation. A number of small vessels were torn; hemorrhage or serous effusion might occur into the sac, which led to tension, nerve disturbance, inflammation, and suppuration. If the rent was left open, the case did much better, as the fluid flowed into the peritoneum and was rapidly absorbed. Tapping of cysts should not be resorted to, the escape of fluid in some cases leading to infection of the peritoneum. Ruptured cysts rarely needed drainage if carefully sponged out. He thought that flushing often acted by breaking up septic material into tiny particles, which were unable to defend themselves against the power of the peritoneum. He maintained that efficiently performed oöphorectomies always stopped menstruation. In cases of ovarian disease it was only necessary to remove the troubled ovary or tube—not both. It was sufficient to empty the drainage tube once in twelve hours under the antiseptic method, but to do so every hour was not too often where no antiseptic was used.

Mr. Meredith replied that he did not consider the opening made in the broad ligament to enucleate a cyst was comparable with the peritoneal wound made in the removal of a kidney. Women at the menopause were passing through a critical time, when kidney trouble often developed, which would hinder ready absorption of peritoneal fluid; he was, therefore, inclined to extend the advantages of drainage to them. Flushing out the peritoneum not only checked hemorrhage, but also avoided the undue irritation of the membrane caused by excessive sponging.

For Enteritis in Infants.—

℞.—Tr. nucis vomicæ . . . gtt. iij.
Syrupi krameriæ . . . ℥ss.
Syrupi cydonii . . . ℥ss.
Aquæ . . . . . ℥j.—M.

℞—Potassii bromidi . . . . gr. viij.
Syrup of belladonna . . . ℥ss.
Syrup of peppermint . . ℥ss.
Aquæ . . . . . ℥j.—M.

Sig.—A teaspoonful of each of these to be given every hour alternately.—*L'Union Médical.*

Camphoric Acid in Inflammation of the Mucous Membranes.—MOSLER, an assistant at the Medical Clinic at Greifswald, reports (*Deutsche med. Wochenschr.*) the results of the use of camphoric acid in the various catarrhs. Camphoric acid is an oxidation product prepared by the action of nitric acid on camphor; it crystallizes in colorless needles; it is with difficulty soluble in water, but readily in alcohol and ether; the taste is acid, astringent, and somewhat disagreeable. It is innocuous in small doses, and is only irritant to the gastric mucous membrane in large doses. Among the indications for its internal administration are the night-sweats of phthisis. In all of the seven cases observed by the author the influence was prompt and encouraged further investigation. Fifteen to thirty grains of the powdered, crystalline acid were given at night. Based upon its disinfecting and astringent action, camphoric acid was successfully used in a case of acute cystitis for three weeks (seven and a half grains, thrice daily); the large amounts of pus present in the urine almost entirely disappeared and the subjective condition remained satisfactory. Milder attacks of catarrh of the larynx and of the nose submitted after one to two weeks' application with a brush and a spray of one-half to one per cent. solutions (at first made with glycerin, but subsequently in bicarbonate of sodium). Inhalations of the one per cent. alkaline solution in phthisis and chronic bronchitis were followed by diminution of the irritation and expectoration, and rendered the expectoration easier. Washing out the bladder with a twenty per cent. alcoholic solution, diluted with lukewarm water to a half to one per cent., answered admirably in chronic cystitis.—*Wiener medizin. Presse*, No. 42, 1888.

Creolin.—The utility of creolin as an all-round remedy is summed up by Dr. Lichtwitz as follows: It is an antiseptic of high powers, a disinfectant, hemostatic, and non-poisonous, as large a dose as 120 grains having been taken with no bad results. In cases of tænia solium and oxyuris vermicularis, in 15 grain doses in capsules, it has proved beneficial; and in parasitic skin troubles and eczema, given in soap liniments, its action is especially efficacious. Internally it is recommended for all troubles of the digestive tube accompanied by fermentative processes, and when the stomach is the seat of cancer. In diseases of the larynx and trachea it may be given with advantage by inhalations, or the powder applied directly to the morbid tissues. There are many cases of ulcerative keratitis which it alleviates, but some in which it has no appreciable effect.—*Les Nouveaux Remèdes*, October, 1888.

Chromic Acid in Excessive Sweating.—A circular has been sent to all the Prussian Army medical officers, advocating chromic acid as an economical and efficient means of checking excessive perspiration. In hyperhidrosis of the feet the application of a ten per cent. solution, repeated every three or six weeks, is sufficient to prevent any inconvenience from this source.—*Medical Press*, October 24, 1888.

Impetigo.—The contagious nature of impetigo induces M. ELOY to insist upon the isolation of patients affected with it, and where this is impracticable, to have towels and brushes specially set aside for their use. The patient should bathe frequently in antiseptic water and apply the following ointment:

℞.—Boric acid . . . . . gr. xv.
Vigo's ointment . . . . ℈iv.
Vaseline . . . . . ℥j.—M.

To be spread on fine linen and applied to the diseased surface.

Successful Splenectomy.—A successful splenectomy was performed on August 2d last, at the Charity Hospital, New Orleans, by DR. G. B. LAWRASON. The patient was a woman, aged thirty-one years, who had been under treatment for uterine fibroids. She presented a large tumor of the abdomen to the left of the median line, which was diagnosticated as an enlarged spleen. It was entirely unconnected with the uterus, and was very movable, and could be pushed upward under the left ribs, and as there seemed to be periodicity in the paroxysms of pain large doses of quinine were ordered to prevent them. The pains becoming more continuous, hypodermatic injections of strychnine and codeia were made twice daily in the spleen.

The patient obtained no benefit from the treatment and strongly begged to have the tumor removed. After some hesitation, on account of the great pain which it was causing and the comparatively favorable prognosis from the fact of its being a floating spleen, extirpation was decided upon. On August 2d the patient was chloroformed, and with the assistance of Drs. Souchon and Logan and members of the Hospital staff, the operation was performed. An incision was made in the median line, extending from about an inch above the umbilicus toward the symphysis, about seven inches in length. The spleen was found under the incision and removed by slipping it sideways. The pedicle proved to be about eight inches in length, and was very much twisted. The return circulation was so impeded that the veins were dilated to the size of intestines. Two ligatures were applied about

an inch apart and the pedicle divided between them, leaving a stump about six inches in length. The arteries and veins were tied separately in the stump as an additional safeguard against hemorrhage. No blood was lost, so the peritoneal cavity was closed immediateiy with fourteen sutures. The spleen weighed 54¼ ounces, measured 12½ inches in length, 7 inches transversely, and almost 4 inches at its thickest point.

On being drained of its blood it shrank nearly to normal size. On microscopic examination nothing abnormal was found. The recovery of the patient was uninterrupted until the 14th, the temperature not having risen above 99⅜°. On that date she had a severe chill, the temperature rose to 101°, and there was great pain referred to the left side. This was all easily controlled by quinine. On the 16th the patient was allowed to get up, the temperature and pulse were normal, and the appetite very good, but not exaggerated. The patient returned home the first of October, feeling perfectly well. During the operation after opening the abdominal cavity the pulse became very weak and rapid, but after removing the spleen it resumed its normal strength immediately. During the treatment three hypodermatic injections were given to produce rest; each time an abscess was formed at the seat of puncture.—*New Orleans Medical and Surgical Journal*, November, 1888.

For the Green Diarrhœa of Children.—For the diarrhœas of children, particularly for the green, undigested evacuations containing casein particles, ZINNIS (*Union Méd.*) enjoins the withdrawal of all starchy food with the following prescription:

℞.—Aquæ fœnic. . . . . 75 parts.
Liquor. calcis . . . . 6 "
Bismuthi subnitrat. . . . 3 "
Syrup. aurantii flor. . . . 15 "—M.

S.—A dessertspoonful every two hours.—*Deutsche med. Wochenschrift*, September 6, 1888.

Management of Cerebral Hemorrhage.—DR. LIÉGEOIS treats light attacks by absolute rest in bed in a shaded room, the head of the patient being elevated and all constriction removed. If the case is severe, he bleeds at the arm, whether the pulse is free or weak, and gives, later on, iodide of potassium internally. Counter-irritation in the form of sinapisms and vesicants, is applied to the chest and extremities, and cold dressings are used on the head every three or four hours. If the onset of the disease has followed a hearty meal, he gives an emetic of ipecacuanha.

When internal stimulation is desired, Hoffman's anodyne, ammonia, or ether, hypodermatically, are found the most reliable agents. For the two weeks following the apoplexy he regulates the diet, and if paralysis results he treats it by massage and electricity. For the more remote effects he relies on the iodide of lithium or sodium, and as the action of these drugs is debilitating to some patients, he adds a tonic of arsenious acid.—*Revue Générale de Chirurgie et de Thérapeutique.*

Intermittent Hemoptysis in Phthisical Subjects.—DR. J. LOWENTHAL, of Frankfurt on Main, in No. 39 of the *Centralbl. für klin. Med.*, describes two cases of intermittent hemoptysis in phthisical patients, one of which had already been reported by Silber-Schmidt. In the first case there was no hereditary predisposition. There were dulness at the left apex and a cavern at the right. The sputum contained large numbers of bacilli tuberculosis. The liver and spleen were not enlarged. There were night-sweats and intermittent fever. On May 27th, at noon, an attack of cough and blood-spitting set in, some ten ounces of blood being lost. On June 1st, at about noon, two pints of blood were lost, and on June 2d there were several minor attacks, in which about one pint was lost. On June 3d, twelve ounces were lost. The spleen was now found to be perceptibly enlarged. From 10 A. M., June 6th, to 11 A. M., June 7th, seven ounces of blood were lost. The spleen was yet enlarged. From 10 A. M., June 10th, slight hemoptysis continued. At this stage, Gerhardt made the diagnosis of intermittent hemoptysis and ordered quinine, fifteen grains, morning and evening, until June 20th, during which time no blood appeared in the expectoration. On June 20th, the spleen was no longer enlarged.

The second case was that of a man who had already had bloody expectoration. Creasote was administered, improving the general condition. From July 14th to 17th, at intervals of eighteen hours, the patient spat blood. The spleen was somewhat enlarged. Upon the administration of quinine the expectoration of blood ceased, and on July 26th, the spleen was no longer enlarged. The sputum contained bacilli. From the regularity of interval of the recurring attack, from the enlargement of the spleen, and from the efficacy of quinine, the author diagnosticated a masked intermittent. He explains the occurrence of the intermittent hemoptysis by the fact that in cases of masked intermittent the concentration of the malarial poison is not sufficient to cause a typical attack of fever, but that the poison operates upon some point, the normal resistance of which is diminished. The progress of the disease is attended with erosion of a vessel, which ruptures from the increased blood pressure caused by the influence of the malarial poison.—*Wiener medizin. Presse*, No. 42, 1888.

Phenic Acid in Skin Diseases.—The internal use of phenic acid in pruriginous affections is highly thought of by DR. AUGAGNEUR, and is especially efficient in eczema and psoriasis. He gives it in the following combination:

℞.—Phenic acid crystals . . . gr. vij.
Syr. aurantii . . . . ℥vj.
M. Glycerine q. s. to dissolve acid.

The dose for an adult is two teaspoonfuls daily.—*Revue Générale de Clinique et de Therapeutique.*

Balsam of Peru for Idiopathic Leukoplaques and other Disorders of the Buccal Mucous Membrane.—Many remedies, both medical and surgical, have been used in the treatment of the so-called idiopathic leukoplaques or psoriasis of the buccal cavity. The use of the galvano- and thermo-cauteries and the sharp curette are rational enough, but the condition is apt to recur. Medicinally, nitrate of silver has been applied locally, and alkaline mouth washes have been used. Corrosive sublimate is efficient only in cases of specific origin. Chromic acid, tincture of iodine, iodoform-ether, and iodoform-alcohol are unreliable. Lactic acid is an excellent remedy, but it has the disadvantage of injuring the teeth, no matter

how carefully applied. The same objection applies to salicylate of sodium. Recently, papayotin, in five to ten per cent. solutions, has been recommended; but the preparation is often unreliable, decomposes readily, is expensive, and only exercises a pain-relieving influence and forms a covering for the denuded surfaces, not influencing the disease process. After numerous trials, ROSENBERG (*Therapeutische Monatshefte*, October, 1888) recommends the balsam of Peru applied with a brush. Two points should be borne in mind: the remedy must be used for a long period; and the preparation must be a good one. The application is followed by slight burning and increased salivary secretion, and is made one to three times daily by the patient himself. It is also thought that in mercurial stomatitis this method of treatment not only diminishes the pain but also the salivation, and that it shortens the duration of the disease. There seems to be no reason why the utility of balsam of Peru should be confined to affections of the mucous membrane of the mouth. The remedy should also prove useful in disorders of other mucous membranes, particularly of those of the urethra, the rectum, and the female genito-urinary tract. The author concludes as follows:

1. Balsam of Peru has an analgesic influence upon painful conditions of the buccal mucous membrane dependent on tissue changes.
2. It hastens granulation and repair of lost tissue.
3. It causes the disappearance of epithelial opacities, in case of old leukoplaques, however, often only after protracted treatment.

When to take Medicines.—Alkaline medicaments should be given before meals. Iodine and its preparations should be given during fasting, when they become rapidly absorbed in their own forms and do not undergo the changes caused by the presence in the stomach of food acids and starchy materials. Acids are best taken midway between meals, when they become rapidly diffused. If, however, it is desired to limit the production of gastric juice, they are given just preceding a meal. Arsenic, copper, and like irritants come after meals, likewise, cod-liver oil, phosphates, and malt preparations. —*Journal de Méd. de Paris.*

Chloroform Water as a Hæmostatic.—ULBRICH (*Präg. med. Wochenschrift*, No. 32) recommends chloroform water (1 : 10 well shaken together) as an effective hæmostatic in bleeding from the gums after the extraction of teeth. Chloroform water is not only a hæmostatic, but also an antiseptic and anodyne in painful and mycotic affections of the mouth. It has also been used for the severe stomach pains of neurasthenia.

℞.—Aquæ chloroformi . . . 100 parts.
Aquæ aurantii corticis,
Aquæ menthæ piper. . āā.50 parts.—M.

S.—Take tablespoonful, three times a day.—*Deutsche med. Wochenschrift*, September 6, 1888.

The Results of the Surgical Treatment of Diffuse Purulent Peritonitis and of Peritoneal Sepsis.—WITZELL, of Bonn, before the Surgical Section of the Sixty-first Congress of Naturalists, discussed this subject:

Though the peritoneum, under certain circumstances, bears well the presence of extremely irritating, infectious matters (as in the reposition of the injured and soiled intestine), although surgical operations for diffuse peritonitis are successful, sometimes in spite of doubtful cleansing of the abdominal cavity, still the method of operation in peritonitis must be improved in the direction of thoroughly, and with safety, cleansing and disinfecting the cavity. The methods hitherto used (removing the intestines or filling the abdominal cavity with antiseptic fluid, evacuated by turning the patient on his side) expose the patient to severe shock without sufficient warrant of entire success. As a result of numerous experiments with animals, Witzel recommends making numerous perforations in the peritoneum, the introduction of long drainage tubes in various directions and passing through these a warm solution of common salt. That this method is also capable of removing the products of inflammation was demonstrated by three cases thus treated—demonstrated at the autopsy, it is true, for, on account of the severity and duration of the existing condition, the operation was powerless to prevent death. It is important to distinguish between peritoneal sepsis and diffuse peritonitis. In the former an enormous development of septic irritant matter occurs in a cavity, the absorbing power of which remains unchanged; the septic products are not prevented entering the system through the large lymph spaces. In purulent peritonitis, however, there exists an inflammatory condition of the serosa, and the lymph spaces are early occluded. Thus, in peritoneal sepsis, therapeutics is hopeless. In the transition forms of purulent-putrid peritonitis, the degree of general infection determines the result. Some successes have been had from the surgical treatment of such cases (Krönlein, Mikulicz, etc.); also, without finding and suturing the perforations in the stomach or intestine has a cure been effected by incision and drainage (Lücke). In true purulent peritonitis, not the result of perforation, we have in its progress, so to speak, to do with a more or less simple abscess; for this reason, surgical measures promise much better results. The progress is equally favorable in cases of so-called idiopathic and in true puerperal peritonitis; in metastatic puerperal pyæmia temporary relief, at least, may be secured by evacuation of the large exudate. In the discussion, Krönlein reported that he had operated in six cases of diffuse purulent-putrid peritonitis, with success in but one. He prefers the median incision and disinfection from this point to multiple incisions.—*Berlin. klin. Wochenschr.*, Oct. 15, 1888.

Pastels for the Sick-room.—

℞.—Iodine . . . . . 1 part.
Acidi salicylic. . . . . 2 parts.
Paraffine . . . . . q. s.

These ingredients are mixed, a wick supplied, and then burned at a candle. The salicylic acid is changed into phenic, and the iodine set free in its own state.—*Revue de Thérapeutique.*

Acne.—For two years LASSAR has successfully treated acne by the application of an ointment of naphthol, 10 in 100. This is washed off in an hour and reapplied on the second day, when desquamation occurs without any cicatrix remaining. In more obstinate cases a camphor salve is also added.—*Lyons Médicale.*

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MENINGITIS IN TYPHOID FEVER.

THE more closely the clinical history of typhoid fever is studied, the more important do its complications appear. Betke, from an analysis of a series of 1420 cases in the hospital at Basle, found only 672 free from complications, whereas 748, or 52 per cent., showed complications of a more or less marked character. It is obvious that no hard and fast line can be drawn between the complications of an acute febrile disease and the mere intensification of certain processes with corresponding prominence of local symptoms. We agree with those who place intestinal hemorrhage, perforation, and peritonitis among the complications. To the same category it seems proper to refer such results of parenchymatous degeneration as atrophy of muscles, abscess of muscles, parotitis, and nephritis. The category to which hypostasis, œdema, thrombosis, embolism, and infarction, with their results, should be referred is a matter of opinion. Whether these processes should be regarded as belonging to the primary disease or as complications of it, would depend, to a great extent, upon the prominence of the morbid phenomena to which they give rise. On the other hand, pneumonia, erysipelas, phlegmonous abscesses, diphtheria, and other septic processes are obvious complications, concerning the relation of which to the original disease there can be no question.

Among the less common of this latter group is meningitis, the occurrence of which as a complication has, from time to time, been the subject of much discussion in view of the fact that, while symptoms pointing to its existence are by no means rare in the course of typhoid fever, for a long time its anatomical lesions had been sought for in vain, even in instances in which the symptoms had been during life pronounced. More recently, the occurrence of cases characterized by convulsions, paralysis, hyperæsthesia, opisthotonos, with the discovery upon examination, *post mortem*, of the associated lesions of cerebro-spinal meningitis, has placed this affection indisputably among the complications of typhoid.

FREYHAN (*Deutsche med. Wochenschr.*, August 2, 1888) has recently reported three cases of meningitis occurring as a complication of typhoid, observed in the wards of Dr. Fürbringer, in the Hospital Friedrichschain, in Berlin. These cases occurred in individuals aged nine, twenty, and twenty-six years respectively. They all terminated in recovery; nevertheless, the symptoms were so well marked as to leave no doubt of the propriety of the diagnosis. As the result of the study of these cases and of the literature of the subject, which is limited, Freyhan makes some general statements, which are of value as illustrating the clinical history of this complication and as shedding light upon the differential diagnosis between mere functional disturbances of the nervous system and meningitis.

The time of the occurrence of meningitis as a complication is in the second period of typhoid fever, occasionally not until the convalescence. Prodromes are, as a rule, absent; occasionally the outbreak is preceded by headache and restlessness. The earlier symptoms are severe headache and vomiting. Headache occurring after the end of the second week thus becomes significant. It is often diffuse, most intense in the occipital region, sometimes localized in the brow, continuous with occasional intensification. It is associated with pains in the neck and back and tenderness upon pressure along the spine; there are also muscular stiffness and opisthotonos. Intellectual disturbances range from mere confusion to furious delirium or deep coma. Most significant symptoms are paresis and paralysis. They vary in intensity from mere weakness of a leg or arm difficult to recognize, to complete hemiplegia. Partial palsies of the facial or oculo-motor nerves and paralysis of the tongue likewise occur. Especially characteristic is the rapid development of optic neuritis. Paralysis of spinal nerves is less common, but hyperæsthesia

and increased cutaneous and tendon reflexes are especially important. The fever is of an irregular remittent type.

The differential diagnosis between meningitis as a complication and mere intensification of the symptoms of meningeal irritation in uncomplicated typhoid will rest chiefly upon the association of a number of symptoms pointing to meningitis, and especially upon the simultaneous occurrence of hyperæsthesia, intensification of the reflexes, and positive motor disturbances. The distinction between meningitis of the ordinary form and tubercular meningitis is not always practicable. The differential diagnosis between meningitis and other forms of nervous disease occurring as complications of typhoid fever, such as hemiplegia, paraplegia and monoplegia due to central local lesions, and peripheral neuritis, rests upon well-recognized data. These affections almost invariably occur during the period of convalescence, and are not accompanied by fever or marked cerebral symptoms.

As regards the etiology of meningitis as a complication of typhoid fever, two views may be advanced: first, the doctrine of a localization of the typhoid infection upon the meninges, and, second, that of a mixed infection. Freyhan inclines to the former of these views. In the present state of knowledge this question cannot be decided; for our own part, we believe it more in accordance with the natural history of typhoid fever and cerebro-spinal fever to explain their association on the principle of mixed or independent infection.

THERE has been an unusual, and almost epidemic, prevalence of typhoid fever in the eastern or French-Canadian quarter of Montreal. Nearly one-fourth of the beds at the Hotel Dieu and other hospitals are now occupied by cases of fever. The Board of Health, having entered upon a thorough investigation of the epidemic, early discovered that the law requiring physicians to report the contagious cases, including typhoid fever, occurring in their practice, was not heeded by the fraternity of the French quarter. Prosecution of the alleged delinquents was ordered and several were brought into court under arrest. The judge declined to try the cases, however, holding that the health officer had full jurisdiction in the question of the non-reporting of contagious diseases. Under a discreet administration of his powers, by that officer, Dr. Louis Laberge, it is probable that the important duty of a timely notification of diseases tending to become epidemic will be accepted by the physicians of that frequently afflicted city. The interests of the public health call for it, not only, but it is also greatly to the advantage of the medical profession that the onset and course of those diseases shall be fully known. An amicable understanding between the health authorities and the profession in this matter is a consummation greatly to be desired.

CREMATORIES for destruction of garbage and dead animals are not uncommon in our western cities, but the only one we know of in the Atlantic line of States is the one recently built at Coney Island, New York, for the Board of Health of the town of Gravesend, which town has an enormous summer population, and an enormous amount of hotel-waste. The furnace was accepted, after trial in the presence of experts. It is built of brick, and is twenty feet long, eight feet high, and five feet wide. It will destroy four or five tons of wet hotel garbage and a number of dead animals in about an hour. It does its work without odor, except during the process of charging.

A CHICAGO Associated Press dispatch announces the resignation of Dr. N. S. Davis, as editor of the *Journal of the American Medical Association*, to take effect on January 1st next, and that the Trustees of the *Journal* have elected Surgeon-General John B. Hamilton, of the U. S. Marine-Hospital Service, to fill the position thus vacated.

DR. JOSEPH O'DWYER, the originator of intubation of the larynx, has been appointed Professor of Diseases of Children in the New York Post-Graduate Medical School and Hospital.

DR. R. J. HALL has been appointed to fill the Chair of Anatomy, at the College of Physicians and Surgeons, New York, made vacant by the death of Dr. Sabine. Dr. Charles McBurney has been made Adjunct Lecturer on Surgery.

THE book of Sir Morell Mackenzie, which is said to have reached the enormous sale of 100,000 copies in two weeks, it is stated, is approved by the Empress-Queen Frederick, who has sent him an autograph letter expressing her complete approval of the manner in which he has performed his difficult task, and confirming some of his statements by her own independent knowledge.

THE Third Congress of Russian Physicians will be held at St. Petersburg in 1889; an exhibition, embracing the various departments of medicine, will be held in connection therewith.

A NEW institute for the practical study of sanitary science has been organized at Rome, being the tenth school of this kind to be undertaken in Europe. Original research in sanitary subjects will receive governmental aid through the Italian National Board of Public Health. The curriculum promises to be very thorough and the school will have authority to grant a diploma to those who successfully pursue a full course of study. The institute will be under the direction of the eminent Dr. Paglioni, now the Professor of Hygiene at the University of Turin, who will also occupy the chair of sanitary engineering.

A PRIZE of five thousand roubles is offered in Russia for the best research on the development of toxic elements in uncooked salt fish, to be handed in to the Minister of Interior by January 1, 1893. The scheme to be filled is as follows:

1. To define, by exact experimentation, the physical and chemical nature of the poison.
2. To study, on animals, its effect on the circulatory, digestive, and nervous systems.
3. To determine its rapidity of absorption.
4. To explain the signs by which affected fish may be distinguished from non-affected.
5. To describe means which will prevent the development of poison in fish.
6. To indicate the antagonists to the toxic elements and the medical measures to be employed in cases of persons poisoned by them.

MISS FLORENCE NIGHTINGALE is said, by an English publication, to be a confirmed invalid from a spinal disease incurred through over-devotion to the cause of nursing. She went home sick from the Crimea, and her health was never thereafter thoroughly reëstablished. She is now approaching her seventieth year, and is destined to pass the evening of her days as an inmate, and its most favored inmate, of St. Thomas's Hospital, London. It was at that institution, in 1858, was established the Nightingale Fund of £50,000, in perpetual commemoration of the heroic labors of that lady in the Crimean hospitals.

REVIEWS.

HYGIENE OF THE NURSERY. By LOUIS STARR, M.D., Clinical Professor of the Diseases of Children in the Hospital of the University of Pennsylvania, etc. 8vo. pp. 202. Philadelphia: P. Blakiston, Son & Co., 1888.

THIS book contains much that will be of value to young mothers, for whose guidance it has been written. The chapter on dietetics is full and explicit, as are also the directions as to the clothing and the ventilation and character of the nursery. The term "scarlatinal nephritis" is perhaps somewhat too technical for the lay reader, and the description of the lingual papillæ is unnecessarily accurate.

We do not think that the majority of the profession will agree with the author in his statement that hand-fed babies are stronger and enjoy better health than those nourished at the breast, not only because of the practical difficulties of good hand feeding, but also because the tendency toward such a course is already so unfortunately common among women that the profession should not only encourage maternal care, but, of all things, refrain from making such statements in books prepared for the popular direction of the laity.

In the chapter on disinfectants we are surprised to find copperas, sulphate of zinc, and carbolic acid are the disinfectants recommended. Not only are these not the best disinfectants, but it is also a fact that the poisonous properties of corrosive sublimate cannot be the explanation for its non-appearance on the list since carbolic acid is far more deadly. The table of the eruptive fevers as to their date of incubation, etc., is well condensed, but of doubtful utility.

The book, as a whole, is a safe and reliable guide, pleasantly written and presented in attractive form, but scarcely adapted to the perusal of "my little patients, some of whom, in the rapid passing of time, may soon assume parental duties," to whom "this volume is affectionately dedicated."

SOCIETY PROCEEDINGS.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, October 22, 1888.

THE VICE-PRESIDENT, CHARLES K. MILLS, M.D., IN THE CHAIR.

DR. WILLIAM OSLER presented a

NOTE ON PACHYMENINGITIS HEMORRHAGICA.

There are several points of interest in this condition:

First. Is it in any sense of the term an inflammatory process? Practically, we see one of three conditions in these cases:

a. Subdural vascular membranes, often of extreme delicacy.

b. Simple subdural hemorrhage.

c. Combination of the two—vascular membrane and blood-clot.

In two specimens which I showed last year at the Pathological Society, I remarked on the absence of any of the features to which we could apply the term inflammatory.

Dr. Joseph Wiglesworth, in the January number of the *Journal of Mental Science* for this year, takes the same ground. Apart from injuries, dura-arachnitis is rare. I have seen two instances in the specific fevers. In both there was delicate fibrinous exudate between the dura and the arachnoid.

Second: Which is antecedent, vascular membrane or hemorrhage?

It is usually stated that the hemorrhage occurs first, and from the blood-clot, when organized, the vascular sheet arises. This may be the case, and the fact that subdural hemorrhage is often found alone—in fifteen out of forty-two cases in Wiglesworth's series—lends weight to this view. On the other hand, the vascular membrane may exist without a trace of past hemorrhage; neither staining nor melanin grains, simply a fibrous sheet, varying in thickness and permeated with large vessels. The specimen here shown illustrates this condition: It was removed from a man, aged fifty-six, who died of aneurism. The skull cap was unusually thick and dense and extremely vascular. The meningeal arteries ran in deep grooves through the bone. There were many points of vascular union between the inner table of the skull and the dura. In spite of this, the calvaria was removed without much difficulty. The inner surface of the dura was everywhere covered with a fibrous, highly vascular membrane. Toward the vault it was from half a line to a line and one-half in thickness. Toward the base it was extremely thin. It was chiefly made up of large veins forming in places beautiful arborescent tufts. On section, where the membrane was thickest, the veins bled freely, but there was no trace of clot. Microscopically, the membrane was composed of bundles of connective tissue forming a supporting framework for the numerous vessels. At one small area alone, in the middle fossa on the left side, there was a brownish-red staining on the surface of the vascular membrane.

In the two specimens which I studied last winter the identical condition existed, though in neither was the blood-supply abundant, nor the vascular membrane thickened.

Third. Whence the hemorrhage, dural or pial?

I have always thought the former, but I see that Wiglesworth, in referring to the atrophy of the convolutions as a possible cause for hemorrhage, speaks thus: "The pia mater over a variable area may be so intensely congested as actually to resemble an ecchymosis. . . . It is manifest that the conditions may be highly favorable to actual rupture, and I doubt not that this frequently occurs."

Certainly in the instances I have seen the subdural membranes were intimately associated with the dural vessels, and it seems more probable (and is, I believe, generally acknowledged) that from these the hemorrhage always proceeds.

Fourth. The explanation of the occurrence of this singular structure is by no means clear. The cases are commonly met with in asylums. In general hospitals years may pass without seeing an instance. During eight years in the post-mortem room of the General Hospital of Montreal, no instance occurred. The first specimen which I saw, after having been for fourteen years interested in morbid anatomy, was demonstrated by Virchow, in 1884, at the Pathological Institute. At the Philadelphia Hospital cases are by no means uncommon. Within the past two or three months there have been four specimens found, usually in the bodies of persons from the insane department, but in the three instances to which I have referred, the patients came from the medical wards, and had not shown any mental symptoms.

One reason urged why the subdural hemorrhage is much more common in the insane, is the atrophy of the convolutions so constantly associated with this condition. But there must be something more than this, for if atrophy alone is the chief factor, we certainly would meet with it in phthisis and in other cachectic conditions in which the cerebral wasting is quite as common as in general paresis.

The frequency of this condition in asylum work may be gathered from the fact that Wiglesworth's paper is based upon forty-two specimens which occurred in a series of four hundred unselected post-mortem examinations in which the persons died of various forms of insanity. Wiglesworth's contribution is one of the most interesting and valuable that has been made upon the subject.

DR. F. X. DERCUM asked if Dr. Osler accepted the view of Wiglesworth that this condition of the dura is really due to hemorrhage from the pia. He could not understand how a hemorrhage from the pia could become so intimately connected with the dura. He had always regarded the formation as dural.

He asked Dr. Osler if he had any explanation to offer for the formation of these curious cysts in old cases of pachymeningitis.

DR. JAMES HENDRIE LLOYD said that Dr. Osler had stated that in the case reported there was aneurism of the aorta. He would ask if there was any connection between the two conditions. Were there any brain symptoms? The idea has prevailed heretofore that we mostly find this condition in connection with brain troubles such as general paresis, trauma, and chronic alcoholism.

DR. CHARLES K. MILLS said that the paper was interesting in connection with a case of a child nearly three of age, with spasms of a peculiar type, for which he advised trephining and the operation was done by Dr. John B. Roberts at the Philadelphia Polyclinic. After the removal of the skull and dura mater, a membrane was found which was easily separated from the pia-arachnoid. It was a vascular membrane, between the dura mater and the pia-arachnoid. Œdema and great vascularity of the pia mater were also present. After the escape of some serous fluid, the brain apparently receded. The history was that a clock had fallen upon the child's head, and the question was whether or not a slight hemorrhage beneath the dura had not taken place, or whether there might not have been the formation of a membrane without the presence of a clot. It may be that this is the explanation of some of the cases of infantile spasm of peculiar type. This child often had from eight to ten attacks a day. The day after the operation it had two slight attacks, but since then none. He had seen a considerable number of cases of pachymeningitis hemorrhagica at the Philadelphia Hospital, some of which he had reported.

DR. OSLER did not see how the hemorrhage could be connected with the pia as Dr. Wiglesworth thought. The cases he had seen had been in medical wards,

and had presented no indications of cerebral trouble. Atrophy of the convolutions does not always coexist. If atrophy were one of the prime factors in the causation of this condition, we would expect to see it more frequently in phthisis and the chronic cachexias, in which the cerebral wasting is often quite as marked as in general paresis. There is often as much thickening of the pia-arachnoid and gelatinous œdema between the convolutions in phthisis as in general paresis, yet, so far as he knows, subdural hemorrhage has not been described in connection with phthisis except in the inmates of lunatic asylums. The condition is rarely met with in general practice. He had himself twelve years' experience in general autopsical work before he saw a case of pachymeningitis hemorrhagica. The first case that he saw was demonstrated by Virchow in 1884, in the Pathological Institute in Berlin. At the Philadelphia Hospital the condition is met with two or three times a month, usually in patients from the Department for the Insane.

DR. OSLER then presented a communication entitled

INJURY TO THE CAUDA EQUINA AND SYPHILOMA OF THE CORD AND CAUDA EQUINA.

which will appear in full in an early issue of THE MEDICAL NEWS.

DR. F. X. DERCUM reported

THREE CASES OF SPINAL ACCESSORY SPASM UNSUCCESSFULLY TREATED BY EXCISION OF THE NERVE.

The following cases were placed on record, not only to confirm the inutility of the treatment by excision of the nerve, but also to provoke discussion.

Case I.—J. F., aged forty years, married, and a gas-fitter by trade, presented himself at the Nervous Dispensary of the University Hospital, with the following history: He had had rheumatism at various times, one attack of which had kept him in bed nine weeks. Eight months after this last attack he had rheumatism of the muscles of the shoulders, and then began to have turning of the head to one side, which, up to the time of his first examination, had lasted a year. The movements were at first slight and painless, as they still are, but about one month after their first appearance the movements became excessively violent, coming on in paroxysms, and interfering with sleep. His wife described these attacks as irritable convulsions. However, they were limited to the neck, and were unattended by loss of consciousness. These excessively severe paroxysms were, later on, merely occasional. Short, sharp, jerky movements were, however, never absent. The sterno-cleido-mastoid of the right side felt hard and contracted, and was evidently the principal muscle at fault. Indeed, in the movement observed by us it appeared to be the only one involved, though in the severe paroxysms described by the patient the other neck muscles, doubtless, played a part.

Some years ago, Dr. H. C. Wood successfully treated one of these cases by severely cauterizing the back of the neck with the hot iron, and acting upon this hint, the Paquelin cautery and antimonial ointment were freely used in the present case, but without relief of the spasm.

Finally, the patient was admitted to the hospital under the care of Dr. Agnew, who excised a portion of the right spinal accessory nerve. The immediate effect of the operation was a disappearance of the spasm, but as this man had left the hospital, in which he remained twenty-five days, the small, jerking, rotatory movement of the head had again appeared, and he could discover little, if any, difference in the character of the movement. Whether any of the more severe paroxysms recurred he was unable to say, as the patient has not since been under observation.

Case II.—E. L., aged fifty-eight years, married, a watchcase-maker, likewise presented himself at the University clinic two years ago. When apparently in good health, he commenced to have slight twitching in the neck. This twitching had gradually become marked, and, at the time of making the note, was present in the form of a severe clonic spasm. There was no history of rheumatism or syphilis.

Examination showed that the left sterno-cleido-mastoid and trapezius muscles were affected. The spasm was slow and irregularly recurring, and was accompanied by pain in the mastoid and occipital regions. It interfered greatly with sleep, the patient being kept awake two or three hours after lying down, Phonation also seemed to be occasionally interfered with.

The patient refusing to be cauterized, various internal remedies were prescribed, all without avail. Finally, he was admitted to the surgical wards, and likewise operated on by Dr. Agnew, who again removed a portion of the nerve. However, within a few hours after recovering from the effects of the ether, we might say almost immediately, the spasm recurred, and it persisted during the entire stay of the patient in the hospital, some three weeks. I should remark, however, that the spasm appeared to be less severe. Certainly, the movements were of less extent.

It is exceedingly probable that in this and the preceding case, other muscles than those supplied by the spinal accessory nerve were involved, and to this, in part at least, the failure is to be ascribed. However, I determined to give the method another trial in the following case, which seemed more favorable.

Case III.—M. K., female, aged fifty-five, a housekeeper, presented herself at the clinic with constantly recurring slow spasm of the right sterno-cleido-mastoid. This, she said, had existed for two years. At its commencement she had been in her usual good health. Examination showed the right sterno-mastoid to be markedly hypertrophied; further, that the right trapezius also participated, but to a slight extent. It could not be determined that any other muscle was, or had at any time been involved. The patient complained also of occipital pain, and pain along the dorsal spine.

After a rather prolonged trial of the cautery, galvanism, and various drugs, the knife was again appealed to, Dr. Ashhurst this time being the operator. Owing to the apparently limited character of the spasm, he was hopeful of a good result. Dr. Ashhurst performed an extremely thorough excision, removing over four inches of the nerve-trunk, including not only the supply to the sterno-mastoid, but also that to the trapezius. Previous to making the excision, the nerve was thoroughly stretched, very marked force being used. After the operation, and during the entire stay of the patient in the hospital, some twenty-one days, no spasm whatever was observed. However, that an inference as regards cure would have been altogether premature, was proved subsequently. The operation was performed on the 2d of June, and in

the early part of the following August he received a letter from the patient, bitterly complaining that the spasm had returned, saying that it was "very bad again." Of the extent of the recurrence he could not speak with certainty.

Whether in these three cases the recurrence of the spasm was due to the spreading of the affection to other muscles, or to the vicarious nerve-supply of the sterno-mastoid and trapezius, is, he thinks, a matter of minor importance. The belief that these spasms are centric in origin, certainly gathers additional force, and the problem that presents itself is, whether the centres affected are spinal or cerebral, and how this differentiation shall be made. Secondly, whether a more radical surgical treatment should be invoked. These were the points to which he invited discussion.

He purposely refrained from speaking of the morphia treatment of these cases, inasmuch as its application to dispensary patients, who can be so loosely controlled, would be of more than doubtful propriety.

DR. CHARLES K. MILLS added another to the list of unsuccessful cases treated by this operation. On the 6th of this month Dr. Roberts operated on a case which had been sent to Dr. Mills by Dr. Tomlinson, of Wilmington. Dr. Roberts cut down on the spinal accessory nerve of the affected side, and, after identifying the nerve by the faradic current, excised one and a half to two inches. Two weeks after the operation the patient left the hospital, the spasm somewhat improved, but certainly not cured. Dr. Mills had had a number of these cases, and some years ago wrote a paper on the subject. Two of the cases reported recovered largely under the use of the actual cautery.

One of the questions which arise in a consideration of these cases is whether some of them are cortical in origin, due to lesion of the centres for conjugate deviation of the head. He believed that such a view could be entertained, but probably not for such a case as he had referred to, or for such as Dr. Dercum had reported. The persistence of the spasm and its tonic character in most cases are against the supposition of cortical lesion.

DR. DERCUM said that the point is, How are we to differentiate between the spinal and cerebral cases? If conjugate deviation of the eyes occurred with the movements, there would be reason for supposing that there was a cortical lesion. It is, however, rare to have this association. It was not present in his case.

As an indication of the utter hopelessness of operations on the muscles and nerves, it would be well to bear in mind that the movement is one in which a large number of muscles are involved. There are nine muscles, in addition to the sterno-mastoid and trapezius, which are called into play in the act of turning the head and neck, besides two others which act as rotators at times.

DR. J. H. LLOYD reported the following

CASE OF ALCOHOLIC MULTIPLE NEURITIS,

which presented such a characteristic clinical picture of a disease which is not common, that he was led to report it in spite of the fact that the pathological study was incomplete.

Margaret M., aged about thirty-five years, was admitted into the nervous ward of the Philadelphia Hospital in May, 1888. She had a family history of phthisis on the father's side. The patient had never been very strong, but had had no sickness except the ordinary diseases of childhood. She was married at seventeen and had had four children, the last labor occurring five years before admission. She denied syphilis, and presented no lesions of it. She also denied excessive drinking, but an inquiry of her friends revealed the habitual use of alcoholics, sometimes in excess, from her childhood. Four weeks before her admission she awoke one morning to find both her feet paretic. For one week preceding this she had had severe pains in both legs.

On examination the following facts were noticed: The patient was anæmic, very despondent in expression, and rather emaciated. She presented double wrist-drop, more marked on the left side, with wasting of the arm muscles, also more marked on the left side. There was paralysis, quite complete, of the anterior leg muscles on both sides, with consequent foot-drop, and wasting. The extensor muscles of the left forearm contracted sluggishly to the faradic and galvanic currents, and showed the serial reactions of degeneration: A. C. C. > C. C. C. The extensors of the feet (muscles especially supplied by the anterior tibial nerves) also reacted sluggishly and showed reactions of degeneration to galvanism. The patellar reflexes were abolished. There were small areas of anæsthesia over the legs and feet. She would believe that a pin was touching the foot when it was touching the leg, and confused the legs. The sensory symptoms were very significant. She suffered from severe burning pain on the soles of the feet, and later in the palms of the hands, causing her to complain. The nerve trunks were acutely sensitive. Pressure over the popliteal and peroneal nerves of the main trunks of the arm was productive of most severe pain. The peroneal nerves especially were sore. The muscles of the leg were extremely sensitive to touch, and those of the calves especially were very tender.

The lungs were normal. The heart presented a faint systolic murmur at the apex; not always heard, and possibly anæmic (?). The pulse was small in volume, very rapid, constantly ranging from 122 to 140, and easily compressible. Her temperature was constantly afebrile, except on two days, when it rose to 101⅘°. For more than half of the time she was under observation (about four weeks) the temperature was rather subnormal, falling as low once as 96⅘°. It was taken in the axilla. The loss of appetite was complete, and the tongue was furred. She slept poorly and her mind wandered. At times she was in a well-marked delusional condition, and was restless always and complaining.

The patient, as she lay in bed, with the wrist-drop and foot-drop, bore a striking resemblance to the picture which Gowers has, in his recent work, of a patient with this disease.

The indications for treatment were especially to relieve pain and sustain the power of a failing heart. It was soon evident that the patient would not respond to treatment. The heart, in particular, never improved in its action. Digitalis was given in the form of tincture, infusion, powder, and poultices of the leaves, but the pulse remained rapid and feeble. A full liquid diet was administered in oft-repeated quantities, but alcohol was not given until toward the last, and then in small doses. It is not necessary to detail all the treatment. Partial relief was obtained from the burning pains in the soles of the feet by lotions of carbolized water; and small cantharidal blisters were used with some benefit over some

of the most painful nerves, as the peroneal. The patient gradually sank from a weak heart and died rather suddenly about the end of the fourth week of her sojourn in the hospital.

He regretted to say that the specimens from this case became decomposed, in some unaccountable way, and were unfit for microscopic study. The brain, cord, and affected nerves, clear to their ramifications in the muscles, were removed, and did not exhibit any marked change on gross examination. It was thought that some parts of the posterior columns were sclerosed; an observation which, he believed, has been made before in some of these cases. The history and observation of the case do not point to an original posterior sclerosis, and it may be a question whether such a change in the cord in a case like the one described could not be secondary to the original nerve lesions. It is also a question whether the obstinately feeble action of the heart in these cases may not be due to changes in the pneumogastric similar to those which occur in the other nerves. The heart was small and the valves normal. The liver was fatty and the kidneys slightly congested and enlarged.

DR. W. T. SHARPLESS reported a case of

EMBOLISM OF THE MIDDLE CEREBRAL ARTERY. LEFT HEMIPLEGIA, ULCERATIVE ENDOCARDITIS, AND LATE GENERAL CONVULSIONS, AND SKIN INFARCTS.

M. D., aged thirty-one years, married (service of Dr. C. K. Mills, at the Philadelphia Hospital), had nothing special in family history. Had been very intemperate for several years, and there was a probable history of syphilis although he denied it. He had acute articular rheumatism last winter which was his only attack. In March last he was suddenly paralyzed on the left side and entered the hospital on April 18th. His condition Aug. 1st, when he came under Dr. Mills's care, was as follows: Left hemiplegia; paralysis marked in the arm, less so in the leg, and not at all in the face; œdema of feet and legs, extending to the knees. He had a very loud double aortic murmur that could be heard as far down the back as the sacrum. Area of cardiac dulness was very much enlarged, as was also that of the liver and spleen. The urine contained albumen in considerable quantity, and later hyaline, granular, and a few epithelial casts were found. In testing his sensations it was found that he could not perceive a light touch as well on the paralyzed side as on the other; but the sense of pain was preserved. Paralyzed muscles responded to the faradic current and showed no reactions of degeneration. His temperature rose to 101° or 102° every evening and regularly fell to normal in the morning.

On Sept. 6th he had two general epileptiform convulsions, and later he had another of a similar character.

On Sept. 15th petechial spots were noticed on his legs and ankles, which increased in size and depth until his death, which occurred Oct. 15th. These spots varied in size from that of a pin's head to a dime and showed no disposition to heal. They were probably skin infarcts.

Autopsy, October 15th: Right middle cerebral artery close to, but just beyond its orgin in the internal carotid, was closed by an evidently old embolus. All branches of the middle cerebral beyond this were thready and cord-like, and could be distinctly seen lying in a bed of yellowish softening, which extended over the island of Reil and the internal surface of all the convolutions which cover in the Sylvian fissure. The cortex of the motor zone, except as just stated, was not softened. A cut through about the middle of the lenticular body revealed softening of the entire breadth of the internal capsule and adjoining lenticular and caudate bodies. This softening was in the middle part of the capsule and did not extend into the anterior and posterior third. Evidently some collateral circulation had been established between cortical branches of the middle and the anterior and posterior cerebral arteries. This was plain in several places where the narrowed and atrophic vessels beyond the embolus again took up their calibre.

The heart was enormously hypertrophied. On the tricuspid and aortic valves were the vegetations of recent ulcerative endocarditis, with a perforation on the septum leading from the left to the right ventricle, just below the aortic ring.

Probably the sequence of events in this case was as follows: First, the abuse of alcohol, and the syphilitic infection; then disease of the aortic valves resulting in their thickening and contraction; next, in March last, the embolus lodging in the middle cerebral artery and producing the hemiplegia, and recently the ulcerative process engrafted on the sclerotic valves.

DR. WILLIAM OSLER said that we are apt to think that the cortical cerebral arteries have not special anastomoses, but an examination of many of these cases of total occlusion, particularly of the middle cerebral artery, shows that a collateral circulation can be established, usually, as in this case, by the enlargement of the anterior cerebral, so that the nutrition is maintained in the central gyri.

NEW YORK NEUROLOGICAL ASSOCIATION.

Stated Meeting, October 2, 1888.

THE PRESIDENT, GEORGE W. JACOBY, M.D., IN THE CHAIR.

DR. B. SACHS presented three cases of

MUSCULAR DYSTROPHIES,

referred to in his paper before the American Neurological Association in Washington. The author referred to the relation between pseudo-muscular hypertrophy and other forms of muscular dystrophies, particularly Erb's form. Erb had stated that the two were identical. Erb's juvenile form consisted in atrophy attacking the upper extremity, particularly the deltoids, the infraspinatus and sometimes the latissimus, the lower extremity being intact. The question was whether pseudo-hypertrophy and Erb's atrophy were distinct or merging types.

Case 1 was twelve and a half years old. The child had suffered from severe fright at ten months. She had never walked well. She had diphtheria when four years old and after this her gait had been worse. When six and a half years old increased size of the legs had been first noticed. The characteristic difficulty in getting u from the floor had been present. There was atrophy commencing in the palmar interossei and affecting the arm and forearm. The thenar and hypothenar eminences had not been involved. There was hypertrophy of the whole lower extremity, but the hypertrophy of the legs had not kept pace with that of the thighs. It was apparent that atrophy of the legs would soon supervene.

The atrophy of the hands had not been present two years ago. The case proved that in the atrophy associated with pseudo-hypertrophy it was not necessary that the shoulder should be first affected as in Erb's form. For the interossei to be attacked first was rare, but only proved that any muscle could be affected by the disease, while in Erb's typical form the hand was exempt to the last.

Case 2 was that of a boy of ten years. There was no history of heredity and no constitutional taint. The patient was a newsboy. He had walked late and had been treated for rickets. He had never been able to run and had been always easily fatigued. The hypertrophy in this case was most marked in the thighs. A peculiarity of the case was the fact that the boy could rise from the floor without climbing upon himself as was customary in the disease and considered by Gowers as a differential test. There was some atrophy of the serratus anticus of the right side. The upper extremities were not affected.

DR. BIRDSALL suggested that the face had a peculiar appearance.

DR. SACHS had not referred to this peculiar puffiness, because, while suggestive, it was not sufficiently characteristic of the Landouzy-Dejerine type. It was possible that it might develop into this appearance.

Case 3 was that of a girl of twelve years. She had walked at nine months. At the age of ten months she had fallen and later had had something the matter with the knee. This was treated with the plaster splint in St. Petersburg and recovered. The circumstance was mentioned because it had been denied that so young a child could be successfully treated with this instrument. At the age of three the child had measles, at four smallpox, at six scarlet fever, and at six and a half typhoid fever. The loss of power in the limbs was not preceded by fever or convulsions. Five months ago it was noticed that motion was impaired in the toes, there was a modified gait, fatigue and difficulty in mounting the stairs. There was no pain. There had been at times a creeping sensation of the skin, and once fibrillar contraction had been noted. The atrophy affected the right leg, thigh, and gluteal region. The electrical reactions were slightly altered, particularly in the tibialis anticus. For four months the knee-jerk had been absent upon both sides, but now it was exaggerated upon the left side, while upon the right side it was still absent. The gait was that of poliomyelitis affecting the anterior group, while the history was one of progressive muscular atrophy of the peroneal form. The gradual onset without convulsions or fever and the progressive character of the affection and the atrophy which was not so profound, distinguished it from atrophies of spinal origin. On the other hand, however, the exaggerated knee-jerk suggested a possible substratum of spinal implication. The case was an anomalous one of a type seldom seen. The author made the diagnosis of peroneal form of progressive muscular atrophy.

DR. BIRDSALL had been interested in the types shown and was not inclined to question the doctor's diagnosis. The last case was, he thought, allied to the progressive muscular atrophy type, rather than to that of an acute poliomyelitis; he thought, however, that there might be grades between the two, that there was no sharp line of demarcation between them. The former presented the characters of a degeneration, while the latter was an acute inflammatory condition which might involve not only the gray matter but extend to the neighboring tracts. The speaker had a case in mind diagnosticated as acute inflammatory poliomyelitis. There was involvement of the muscles of the anterior and posterior aspects of the thigh, slight affection of the anterior tibial group, and exaggerated reflex showing extension to the lateral column. In the speaker's opinion the different varieties of atrophy represented grades rather than distinct pathological entities. The fact that the atrophy in one case had affected an arm and a leg, in one was hemiplegic, and in another paraplegic, should not blind one to the possibility that the pathological processes might be the same. In the present stage of our histological knowledge, he thought that we were not even able to separate cases into those of myopathic and myelopathic origin. The number of cases was meagre and our methods of examination were not calculated to reveal minor degrees of atrophy. In the muscles, nerves, and cord it might be possible to have a gradual destruction and regeneration going on which would escape detection. The value of the electric reaction was equally open to doubt. If fibres degenerated one by one, it was probable that the reaction would remain normal until the number of atrophied fibres had attained a certain grade. The degeneration reaction would be, in any case, interfered with by the normal reaction of the healthy fibres. The question was whether there was great uniformity in these various types or great diversity; he predicted the latter, the types running into each other.

DR. PUTNAM JACOBI suggested that the gluteal muscles were unaffected in the second case presented. This accounted for the lack of the usual difficulty in getting up from the floor.

DR. SACHS accepted this explanation. He had referred to the point because Dr. Gowers had mentioned the symptom as pathognomic.

DR. DANA referred to a case of pseudo-hypertrophy in which the disease had advanced from the calves to the thighs, and thence to the shoulder girdle. There was slight hypertrophy of the forearm, the part said to be unaffected in Erb's form. The fundamental distinction between the symptoms of chronic anterior poliomyelitis and progressive muscular atrophy was the retrogressive or stationary character of the former, and the progressive character of the latter. It was difficult to criticise the diagnosis in Dr. Sachs's last case. He would prefer to postpone his decision, but would suggest that a careful examination of the urine be made for lead. A number of cases of anterior poliomyelitis in children had been recently ascribed to lead.

DR. GRAY would, upon *a priori* reasoning, decide the case as one of chronic anterior poliomyelitis, but that anterior horn myelitis was, as had been suggested, retrogressive. Muscular atrophy could be, he thought, divided into that of spinal and peripheral origin; of the latter, however, we must distinguish between that arising in the muscles and that due to peripheral neuritis. The results of treatment varied. There were cases which, with rest in bed, faradism, and gentle massage, improved, while other cases were unaffected by treatment. He thought that the former were of spinal while the lateral were peripheral, either neural or muscular in origin. Formerly the diagnosis of a central or peripheral origin was deter-

mined by the presence or absence of the reaction of degeneration. Now confusion surrounded this test also. He inquired in regard to opinion upon this point. Was the degeneration reaction present only where the muscles were degenerated?

DR. SACHS closed the discussion. He was pleased that Dr. Birdsall, who had not heard his paper in Washington, had been in substantial agreement with it. While there was diversity of types there was no anatomical distinction between pseudo-hypertrophy and atrophy. The primary and secondary affections were distinguished by their symptoms. The primary affection occurred early, there was absence of fibrillary contractions and absence of the reaction of degeneration. This group included pseudo-hypertrophy, Erb's type and that of Landouzy and Dejerine.

DR. GRAY begged to refer to a case recorded by Schultze, which had been assumed to be primary, but in which there had been found changes in the cord.

DR. SACHS explained that the change referred to was independent and unconnected with the muscular symptoms. It was in a part of the cord unconnected with the muscular tract. He suggested that in Dr. Dana's case the atrophy in the arm might have given rise to a fictitious appearance of hypertrophy in the forearm.

DR. DANA added that the muscles had also the peculiar feel of pseudo-hypertrophy.

DR. SACHS did not question the diagnosis. He believed that all present had agreed upon the desirability of getting rid of the present classification upon anatomical distribution of the hypertrophy or atrophy. He had retained the term peroneal form for convenience only.

DR. M. D. FIELD followed with a paper entitled

OTHÆMATOMA OR INSANE EAR.

The title referred to a shrunken, shrivelled condition preceded by a period of active congestion during which the organ was swollen, hot, and very painful. Between the cartilage and the perichondrium there was effused a sero-sanguinolent fluid, which might become purulent. This was absorbed or rupture occurred with healing and the deformity described. The deformity was not uncommon among the insane. It had been attributed to trophic change. The most general opinion had been that othæmatoma was characteristic only of chronic cases, and usually indicated an incurable condition. The question of a traumatic origin had received some attention. It had been remarked that a Greek statue represented a boxer with this deformity. The author could not recall the reference. It had been said to result in boys from the violence of the game of foot-ball. It had also been observed among soldiers. The author had had under his care the case of a professional pugilist who had presented the deformity in question. Upon directing his attention to it, the man had remarked that it had resulted from a blow upon the head, and that it was common in his profession, that he knew as many as a hundred men similarly injured. Arrangements were made with this man to be present at the meeting. He had been unable to come, but had sent substitutes. Several under-sized, meek-looking professional men, pugilists, here appeared, all presenting the deformity. It could be added, that only one had ever been in an asylum. This one had been in for delirium tremens. It was evident that the insane ear could proceed from traumatism; also that it was more common than was commonly supposed.

DR. PETERSEN felt indebted to Dr. Field for the opportunity of examining the cases. He had seen the condition frequently in the insane, and believed it was always due to traumatism. It was more frequent among the insane, probably, on account of the necessity for restraint, also because the insane were apt to injure themselves in many ways. He also believed that the changes in the walls of the vessels in insanity predisposed to the result. Retrograde metamorphosis of the cartilage and perichondrium, like that of senility, was common in general paresis and other varieties of chronic insanity.

DR. PARSONS exhibited a cast of othæmatoma in the recent state, taken some years ago. He had always considered the condition a result of nerve degeneration. The exhibition this evening had, however, demonstrated that it could result from traumatism alone.

DR. DOUGLAS had seen a considerable number of instances of the insane ear, and thought that it had been due to violence in every instance, either from the attendants, other patients, or the sufferer himself.

DR. INGRAM could corroborate this statement. The insane had the habit of rubbing the ears, which might account for some cases. He could add to Dr. Field's record that of a case of recovery after double othæmatoma during an active maniacal attack. With improvement of the mental condition there was improvement in the ear. Upon recovery there was diminution in the size of the ear but no deformity.

DR PETERSEN added that the reference to the Greek statue had been by Fuerstner, *Arch. f. Psych.*, iii.

DR. FIELD, in closing the discussion, referred to the case reported by Dr. Hunn, after the patient had been struck with a broom-stick, the hematoma appeared upon the opposite ear. Dr. Hunn cited the case as an instance in which traumatism could not have been the direct cause.

PHILADELPHIA ACADEMY OF SURGERY.

Stated Meeting, October 1, 1888.

R. J. LEVIS, M.D., IN THE CHAIR.

REMOVAL OF THE VERMIFORM APPENDIX.

DR. NANCREDE exhibited a vermiform appendix which he removed seventeen days ago. The patient is doing well. This was a hyper-acute case. The child thought that she had injured herself, suddenly experiencing acute pain in the right iliac region. There was vomiting followed by profuse purging. The attack was supposed by the parents to be one of cholera morbus. The vomiting and purging lessened, but from the very inception of the attack there was retraction of the thigh. He was called to see the patient forty-eight hours after the onset. The pulse was then 150. There was moderate pain and tenderness in the region where he supposed the appendix should be, but it afterward was shown not to be exactly in this position. There was no impairment of resonance. The right thigh could not be straightened out, although motion could be made in other directions without causing pain. The temperature was a little over 100°. The next morning at twelve o'clock, the temperature was over 101° and there was, in the right iliac fossa, impairment of resonance on light percussion and tympan-

itic resonance on deep percussion. At three P.M. the temperature was between 102° and 103° and the pulse about 160. He sent the patient to the Jefferson Hospital, where Dr. Barton, and Dr. Conner, of Cincinnati, saw her with him. When the peritoneum was opened purulent peritoneal fluid escaped. There was no limitation of the inflammation toward the median line. He did not find the appendix in its usual position, but after opening with the finger one or two foci of pure pus, the appendix was found pointing directly downward and backward from the outer surface of the cæcum, so that there was much difficulty in applying the ligature. A foreign body was felt which was pushed along the appendix until room was made to ligate between the concretion and colon. A little spot of sloughing was found near the middle of the appendix where the end of the foreign body pressed.

In the discussion on this subject at the recent meeting of the American Surgical Association, it was held by some that the median incision was the preferable one in these cases. In this instance it would have been impossible to reach the appendix through a median incision. It has been advised that the whole wound be left open for drainage. If he had done this, the small intestines would certainly have escaped. There was no limiting membrane. He had seen in consultation one case in which there was secondary hemorrhage after the spontaneous opening of a perityphlitic abscess, the blood probably coming from the deep circumflex iliac. Mr. Hutchinson had, he believed, reported a similar case of severe hemorrhage in which he thought that the blood came from this vessel.

DR. T. G. MORTON said that in the case just reported by Dr. Nancrede the symptoms which demanded operation had existed only a few hours, so that very properly the case should be regarded as belonging to the supra-acute variety of perforative appendicitis. It is somewhat difficult, perhaps, to classify the varieties of inflammation, perforation, and abscess of the appendix, but a division according to the history of the supra-acute, acute, and chronic cases, which he suggested some time since, appears for all practical and clinical purposes quite sufficient. The incision to reach the appendix should be lateral, not median, indeed by the latter it would often be impossible to reach the appendix, and the belly cavity would often needlessly be opened. From numerous dissections he has found that the appendix is normally placed immediately under a point two inches distant from the right anterior superior spinous process, on a line drawn horizontally from the process toward the median line of the body. If the general peritoneal cavity has become involved, it can be washed out as thoroughly by the lateral as by a median section, while by the former more perfect drainage can be secured.

By a lateral incision either vertical or oblique, the appendix can more readily be reached and dealt with, and in the subsequent treatment sloughs, and suppuration can be discharged through the lateral incision; experience has shown that the wound should be left well open except in the supra-acute cases, while such a course could not be adopted in the acute and chronic cases with a median incision.

The presence of the colon, which is more or less firmly held in position at the bottom of the wound, in the cases in which the wound is kept open, prevents any intestinal protrusion during the wound-healing process. The appendix vermiformis almost constantly contains some matter having the appearance of feces; in thirteen consecutive post-mortems recently made at the Pennsylvania Hospital, the appendix in every instance but one contained such matter.

The diagnosis of appendix inflammation, perforation, and abscess ought generally to be readily made, pain in the cæcal region being about the earliest symptom, often paroxysmal, and increased by pressure; nausea and sometimes vomiting; the occurrence of pus is announced by temperature, pulse, and sweatings; perforation of the appendix is often accompanied by the most excruciating pain; general peritonitis by extension of inflammation and symptoms.

Since abscess generally in the ileo-cæcal region is due to appendix disease, we may safely and promptly make an explorative incision, indeed, any uncertainty of diagnosis had best be cleared up by an early section. The danger in all such cases is greatly increased by delay.

DR. MEARS had had two cases in which he operated, one ten years ago, and the other two years ago. In both he made a lateral incision and evacuated the abscesses. In both cases death followed. The post-mortem examination in the first case showed general peritonitis, constriction of the small intestine by a band, and perforation of the appendix, which contained a number of cranberry seeds.

DR. WEIR, who has written ably upon this subject, concludes that the lateral incision is the best. After the occurrence of general peritonitis the median incision is to be preferred to the lateral. There is no doubt as to the value of the lateral incision, but it occurred to him that the median section may be best in certain cases in which it is necessary to deal with complications.

MONTREAL MEDICO-CHIRURGICAL SOCIETY.

Stated Meeting, October 19, 1888.

THE PRESIDENT, WILLIAM GARDNER, M.D., IN THE CHAIR.

DR. BLACKADER read a paper on

FOUR CASES OF PERITONITIS OCCURRING DURING GESTATION.

Case I.—Mrs. R., æt. thirty-two, in the sixth month of her third pregnancy, was suddenly seized with severe abdominal pain. She had always enjoyed good health with the exception of being the subject of constipation. She had taken some purgative medicine, and during the night was obliged to get up to attend to one of her children and got chilled; an hour later severe pains came on. When seen she was suffering severely, her abdomen was slightly distended and tender; temperature 104°; pulse 140, small and weak. Morphia was administered hypodermatically in quarter-grain doses and hot cataplasms applied. Her pulse rapidly failed and it was soon seen that she was suffering from acute general peritonitis. The os was examined and found to be soft, closed, and with prominent neck. There did not appear to be any special contraction of the uterus. By midnight her pulse was scarcely perceptible, but afterward improved. The pains now assumed a bearing-down character, and twenty minutes after a dead fœtus was born. Vomiting of grumous matter set

in and she expired in a very short time. No post-mortem was allowed.

Case II.—Mrs. S., æt. thirty-three, in her eighth pregnancy, had always enjoyed good health. Toward the end of the eighth month of pregnancy caught cold and suffered an attack of bronchitis causing her to be confined to her bed for a week; while recovering from this, acute parenchymatous glossitis set in, the pyrexia lasting three days. On the fourth day the swelling had almost subsided. The same afternoon Dr. B. was sent for in haste and found patient with pinched features, feeble, quick pulse, and a temperature of 103°. There was considerable tenderness over the lower part of the left abdomen, especially in left groin; there was no distention of the abdomen. Morphia was given hypodermatically. Next day she was somewhat improved and only suffered pain whilst turning to either side. In the evening a live child was born with scarcely any pain and very rapidly. Immediately after the birth her pulse was 120, and temperature 101°. Dr. George Ross was called in consultation and also Dr. Gardner. Two days after her child was born her pulse began to fail rapidly and her condition seemed most serious. Vomiting and hiccough set in, and there was great distention of the abdomen. Arrangements were made for abdominal section, but death took place during the administration of the ether and before the operation had been commenced.

Case III.—Mrs. C., æt. twenty-eight. Dr. B. first saw her June 23, 1885, in the sixth month of her fourth pregnancy, and naturally of a very strong constitution. She was complaining of severe abdominal pain with diarrhœa, which she attributed to getting her feet wet a day or two previously. Pulse was rapid, but her temperature was normal. An anodyne was prescribed, and she was not seen again for forty-eight hours, when she had a rapid pulse (108) and a temperature of 99.8° F.; she complained of pain and tenderness in the abdomen, and was confined to her bed. Next morning she was much worse. She had an anxious expression of countenance, considerable lividity of face and lips, with a temperature of 102.5°, and pulse of 120; tenderness was marked in the right iliac region and extended across the median line; there was slight abdominal distention. That evening she was comfortable. At 2 P. M. labor pains came on and the child was born before Dr. Blackader could reach the patient. There had not been much hemorrhage, but the mother was very pale, her lips were livid and her pulse feeble; the placenta was retained. After waiting a short time the placenta was delivered by passing the hand into the uterus; there was no hemorrhage. Vomiting set in shortly, and she died the same evening at six o'clock. No post-mortem examination was allowed.

Case IV.—Mrs. B., æt. twenty-four, consulted Dr. B. about the end of October, 1887, for frequent and painful micturition. She had been married in August. The symptoms did not yield to remedies, and a vaginal examination was made and the uterus was found anteverted and decidedly enlarged. Absolute rest in bed was insisted on. By the end of December she was up and about and apparently as well as ever. She was not seen again until the middle of February. She then complained of pain in the right iliac fossa and back; there was also a good deal of nausea, a thickly furred tongue and constipation. Pulse and temperature normal; no tenderness on pressure was detected. Salines were administered (March 2d) with partial success. A fortnight later the pain became suddenly much aggravated, apparently after eating heartily; there were much tenderness over the cæcum and slight distention of abdomen. Temperature 102°, pulse 100. Slight nausea but no vomiting. The patient was anxious, restless. On the evening of the 5th Dr. Brune was called in and agreed that there was a local peritonitis. A few days later the symptoms became so serious that the question of abdominal section was discussed. There were considerable distention and great tenderness of the abdomen, especially in the right iliac region, and vomiting was frequent. Pulse weak, and 120–130; temperature 101°. Morphia was given hypodermatically and the symptoms gradually subsided. On the 12th the right parotid gland became inflamed, and then the left gland was likewise affected, but both subsided without suppuration. On the 14th of May the patient was safely delivered of a healthy child, labor was in every respect normal and convalescence was uninterrupted. Since her confinement there have been two slight attacks of abdominal tenderness in the right side, with slight pyrexia—but now the patient is quite well.

Dr. Blackader said that there was no reason why peritonitis should not occur during gestation, while there are reasons why it might even be more frequent at that time. A very small number of cases are recorded in medical literature, and he thought it must be of rare occurrence. No reference is made of peritonitis during gestation in any of the works on obstetrics, and very few cases are reported in medical journals. Dr. Blackader could find only four. Dr. Gow reports a case in the *Edinburgh Medical Journal* for January, 1888, which was given by the reader of the paper in full. The patient died, and no cause for the peritonitis could be found. Dr. Gow refers to one of Sir James Simpson's cases in which also no local cause could be found at the post-mortem. Dr. Matthews Duncan reports two cases of a similar kind, in both of which death took place, and no local cause of the peritonitis could be discovered. One of the patients suffered from enteritis. In not one of the cases cited by Dr. Blackader could the exciting cause of the peritonitis be discovered.

The diagnosis of the cases will lie between peritonitis, concealed hemorrhage, rupture of the uterus, and perhaps rheumatism of the uterus. Dr. Blackader said that he much regretted being unable to procure a post-mortem in his fatal cases, but that he had not the slightest doubt that his diagnosis of peritonitis was correct.

DR. J. C. CAMERON said that he did not see why pregnant women should not have peritonitis; their condition would rather predispose to it than otherwise. The great defect in the paper was the fact that no autopsies were held, and whether the peritonitis was idiopathic or not could only be determined by a post-mortem examination. He himself was rather sceptical as to the occurrence of idiopathic peritonitis. Peritonitis may, in these cases, have been caused by a perforating appendicitis, ruptured cyst or tube, abscess, etc.

DR. HARVEY, of Calcutta, related a case in which postpartum hemorrhage was caused by adhesions of the uterus to the parietal peritoneum, this prevented contraction of the uterus. He saw no reason why pregnant women should be exempt from peritonitis, as the same causes which produce it in the non-pregnant would be found in the pregnant.

DR. T. JOHNSON ALLOWAY said that the pregnant uterus could stand a great deal of injury; he himself thought pregnant women were not so liable to peritonitis as other people. In the cases reported by Dr. Blackader he was of the opinion that if a post-mortem examination had been held a ruptured tube would have been found, or remains of old peritonitis may have lighted up pelvic inflammation.

NEWS ITEMS.

Prevention of the Spread of Smallpox among the Indians in New York and Pennsylvania.—Official information having recently been received by the Secretary of the State Board of Health of Pennsylvania, from the Secretary of the New York State Board, of the existence of smallpox among the Indians of the Catarangus and Allegheny reservations in that State, he at once telegraphed instructions to Dr. J. L. Stewart, of Erie, Medical Inspector to the Board for the lake district, to visit the Cornplanter tribe in Warren County, Pennsylvania, the members of which are in constant communication with those of the New York reservations, and take the necessary means for their protection. The instructions were carried out with commendable promptness. The entire tribe was vaccinated and the chief, Marsh Pierce, promised to forbid his people from crossing the border until the disease had disappeared. He rendered every assistance to the vaccine physician, and extended the thanks of himself and his people to the State Board for its timely action in their behalf.

Dressed Poultry.—The Legislature of Massachusetts, according to *The Prophylactic*, has enacted the following: 1. No poultry, except it be alive, shall be sold or exposed for sale until it has been properly dressed by the removal of the crop and entrails, when containing food. 2. Whoever knowingly sells or exposes for sale poultry contrary to provisions of section 1 of this act shall be punished by a fine of not less than five nor more than fifty dollars for each offence. The Boards of Health in the several cities and towns shall cause the provisions of this act to be enforced in their respective cities.—*Journal of Amer. Med. Assoc.*, Nov. 10, 1888.

OFFICIAL LIST OF CHANGES IN THE STATIONS AND DUTIES OF OFFICERS SERVING IN THE MEDICAL DEPARTMENT, U. S. ARMY, FROM NOVEMBER 6 TO NOVEMBER 12, 1888.

By direction of the President, BASIL NORRIS, *Lieutenant-Colonel and Surgeon* (U. S. Army), and HENRY R. TILTON, *Major and Surgeon* (U. S. Army), are detailed as members of the Army Retiring Board appointed to meet at San Francisco, California, by War Department Order dated October 27, 1888, published in Special Orders No. 253, October 30, 1888, from Headquarters of the Army, vice Colonel and Surgeon Elisha J. Bailey and Captain and Assistant Surgeon John J. Cochran, hereby relieved.—Par. 13, *S. O. 261, A. G. O., Washington*, November 8, 1888

By direction of the Secretary of War, JOSEPH C. BAILY, *Lieutenant-Colonel and Assistant Medical Purveyor*, will transfer at once the public funds for which he is now responsible, to Captain and Medical Storekeeper Henry Johnson, who will assume temporary charge of the medical purveying depot in New York City, retaining charge thereof until relieved in person by Lieutenant-Colonel and Surgeon E. P. Vollum, under his assignment as Acting Assistant Medical Purveyor, to whom Captain Johnson will then transfer the public funds.—Par. 1, *S. O. 257, A. G. O., Washington, D. C.*, November 3, 1888.

MCCLELLAN, ELY, *Major and Surgeon.*—Is relieved from duty at Jefferson Barracks, Missouri, and will report for duty at Chicago, Illinois, as attending surgeon and examiner of recruits.—Par. 15, *S. O. 261, A. G. O., Washington*, November 8, 1888.

BARTHOFF, JOHN H., *Major and Surgeon.*—Is relieved from further duty in the Department of Texas, and will report to the commanding officer at Plattsburgh Barracks, New York.—Par. 15, *S. O. 261, A. G. O., Washington*, November 8, 1888.

EWEN, CLARENCE, *Captain and Assistant Surgeon.*—Is relieved from duty at Fort Sidney, Nebraska, to take effect on his present leave of absence, and will report in person to the commanding officer Madison Barracks, New York, for duty at that post.—Par. 2, *S. O. 258, A. G. O., Washington*, November 5, 1888.

APPEL, DANIEL M., *Captain and Assistant Surgeon.*—Is relieved from duty at Fort Davis, Texas, and will report in person to the commanding officer Fort Sill, Indian Territory, for duty at that post, reporting by letter to the commanding general Department of the Missouri.—Par. 2, *S. O. 258, A. G. O., Washington*, November 5, 1888.

PRICE, CURTIS E., *Captain and Assistant Surgeon.*—Is granted leave of absence for two months, with permission to apply for an extension of ten days.—Par. 2, *S. O. 257, A. G. O., Washington*, November 3, 1888.

WOOD, MARSHALL W., *Captain and Assistant Surgeon.*—Is granted leave of absence for one month, with permission to apply for an extension of ten days.—Par. 2, *S. O. 257, A. G. O., Washington*, November 3, 1888.

ARTHUR, W. H., *Assistant Surgeon.*—Upon being relieved by Acting Assistant Surgeon J. L. Ord, will proceed to comply with Par. 21, S. O. 250 c. s., Headquarters of the Army.—Par. 5, *S. O. 123, Headquarters Department of Arizona, Los Angeles, California*, November 5, 1888.

By direction of the Secretary of War, ALFRED E. BRADLEY, *First Lieutenant and Assistant Surgeon* (recently appointed), will report in person to the commanding officer David's Island, New York, for duty at that depot, reporting by letter to the superintendent of the recruiting service New York City.—Par. 14, *S. O. 260, A. G. O.*, November 7, 1888.

OFFICIAL LIST OF CHANGES IN THE STATIONS AND DUTIES OF THE MEDICAL CORPS OF THE U. S. NAVY, FOR THE WEEK ENDING NOVEMBER 10, 1888.

DICKINSON, D, *Surgeon.*—Ordered to Naval Hospital, Mare Island, California.

BRADLEY, G. P, *Surgeon.*—Detached from Naval Hospital, New York, and ordered to the "Iroquois."

PERSONS, R. C., *Surgeon.*—Detached from Army and Navy Hospital, Hot Springs, Arkansas, and ordered to Naval Hospital, New York.

NORFLECK, ERNEST, *Passed Assistant Surgeon.*—Detached from Naval Hospital, Mare Island, California, and ordered to the "Trenton."

HARRIS, H. N. T., *Assistant Surgeon.*—Detached from Naval Hospital, Mare Island, California, and ordered to the "Kearsarge."

SAYRE, J. S., *Assistant Surgeon.*—Ordered to examination for promotion.

BEYER, H. G., *Passed Assistant Surgeon* —Detached from the "Trenton," and granted six months' leave, with permission to leave the United States.

BRYANT, PATRICK H.—Commissioned Assistant Surgeon, United States Navy.

VON WEDEKIND, LUTHER L.—Commissioned Assistant Surgeon, United States Navy.

THE MEDICAL NEWS *will be pleased to receive early intelligence of local events of general medical interest, or of matters which it is desirable to bring to the notice of the profession.*

Local papers containing reports or news items should be marked.

Letters, whether written for publication or private information, must be authenticated by the names and addresses of their writers—of course not necessarily for publication.

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